ARCHIVES OF SURGERY

VOLUME 35 OCTOBER 1937 NUMBER 4

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INCIDENCE OF ASYMPTOMATIC PATHOLOGIC CONDITIONS OF THE APPENDIX

BASED ON A STUDY OF 2,065 CONSECUTIVE INCIDENTAL APPENDECTOMIES

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This paper is based on a study of the records of 2,065 consecutive incidental appendectomies performed at St. Luke's Hospital in the period between 1925 and 1935 inclusive. The word incidental as used here refers to those appendectomies performed during abdominal operations, the indications for which were in no way associated with the appendix. None is included which was performed when present or past disease of the appendix was suspected before operation. Doubtless some of the patients may have had appendical symptoms at some time in the past, but neither the history nor the physical findings suggested any pathologic condition of the appendix.

Originally this analysis was undertaken to obtain a standard for comparison with the pathologic conditions found in appendixes removed because of symptoms attributed to chronic appendicitis. By selecting the material as previously noted and then eliminating all in which any inflammatory condition was present elsewhere in the abdomen, the resultant figures should approximate closely the expected incidence of pathologic changes in the appendix when it is not the cause of the patient's symptoms. In obtaining the set of figures just described, other statistics were obtained which appeared to be of considerable interest. This paper was written to present these figures.

So far as possible, everything was excluded which pertained to the primary operation, and attention was centered on the results of the study of the appendixes. In order to do this, the incidences were calculated on the total number of appendixes in each group under consideration at the time and not on the total number in the whole series. In this way comparisons have been made in which the effect of the indications for the primary operations were largely, if not entirely, eliminated.

Presented before the New York Academy of Medicine, Section of Surgery, Dec. 4, 1936.

REGION OF THE PRIMARY OPERATION

The data in table 1 are given only to show how the primary operations were distributed in regard to the operative region. A detailed list of the operations was felt to be too complicated, particularly as from 2 to 6 different operative procedures other than appendectomy were performed on many of the patients. There is an apparent increase of 110 operations over the total of 2,065, because in some instances the primary operative procedures were done on the same patient in two different regions. Additional operative procedures outside of the abdomen, of which there were a considerable number, were not listed.

The mortality for each group did not appear to be above the normal expectancy, with but one exception. This was for the operations performed for some condition associated with the colon. A careful study of this material and of the causes of death shows that the mortality was not above the normal expectancy, although this does not obviate the fact that it was unwise to add an appendectomy to the primary operation, which was imperative.

TABLE 1.—*Distribution of Primary Operations According to the Operative Region, With Mortality for Each Group* *

Operative Region	Number of Operations	Number of Deaths	Mortality, Percentage
Female generative organs	1,654	13	0.79
Gallbladder and liver	312	5	1.61
Hernia	68	0	0.00
Stomach and duodenum	53	2	3.77
Colon	17	5	29.82
Small intestine	6	0	0.00
Other intra-abdominal regions	65	2	3.07

* The total number of primary operations is increased to 2,175 because often a "primary" operation was done in more than one region on the same patient. When more than one operative procedure was performed in one region they are listed as one operation. Additional operative procedures not involving the abdominal cavity are not listed.

INCIDENCE OF PATHOLOGIC CONDITIONS IN THE APPENDIX

Table 2 lists the pathologic conditions in the appendixes, their incidence (calculated on 1,904 examined microscopically) and the mortality for each group.

No microscopic examination was made of 161 appendixes. Of those examined, 14 showed conditions other than a nonspecific inflammation. These, together with 7 which showed some pathologic process in addition to a nonspecific inflammation, will be considered in tables 13 and 14. The latter 7 are also listed in their proper place according to the inflammatory change.

This leaves 1,890 appendixes which formed the basis for the figures in tables 3 to 12 and in charts 1 to 16. A brief definition of the various types of inflammatory change is given. They have been listed

separately throughout the tables, as it was felt that the term chronic appendicitis was too ambiguous.

No Definite Evidence of Inflammation.—In 702 appendixes no definite evidence of inflammation was found. These made up 37 per cent of the group now being discussed. They were divided pathologically into the two classes listed here.

Normal Appendixes: All appendixes which appeared normal or relatively normal with no definite pathologic change were listed as normal. There were 629 appendixes, or 33 per cent of the total, in this group.

Atrophic Appendixes: The wall of an atrophic appendix is very thin and shows no definite evidence of inflammation. This condition is due largely to pressure from within the appendix, present over an appreciable period of time. Seventy-three appendixes, 4 per cent of the total, showed this pathologic change.

Definite Inflammatory Changes.—As contrasted to the appendixes with no definite evidence of inflammation, 1,188, or 62 per cent of the 1,890 being discussed, showed definite inflammatory change. These have been divided into two main groups according to the degree of the inflammatory change present.

Simple Chronic Appendicitis: In this group were placed those appendixes showing a definite, but not marked, evidence of chronic inflammation throughout the various parts of the wall or only in the mucosa and submucosa. This lesion was found in 573 appendixes, or 48 per cent of those showing inflammatory change (30 per cent of the total group).

Marked Inflammatory Changes: As contrasted to the foregoing group, a more extensive inflammatory lesion was found in 615 appendixes, or 52 per cent of those showing inflammatory change (32 per cent of the total group). The various types of conditions which this included will be discussed.

Chronic catarrhal appendicitis is characterized by a moderate degree of chronic inflammation in the mucosa and submucosa, with definite catarrhal changes in the mucosa and usually a slight infiltration in the muscle. This lesion was found in 82 appendixes, or 13 per cent of those showing marked changes (4 per cent of the total group).

Chronic exudative appendicitis is a definite pathologic lesion which is often seen as a late stage in the recovery from an attack of acute appendicitis. It is characterized, as the name indicates, by a marked chronic exudative process. There is a dense extensive infiltration with the various cells typical of chronic inflammation, and often chronic edema is present. The lumen of the appendix, the blood vessels and the perivascular tissues frequently contain some polymorphonuclear leukocytes. This lesion was found in 157 appendixes, or 26 per cent of those showing marked changes (8 per cent of the total group).

Acute appendicitis, which requires no definition, was found in 33 appendixes, or 5.4 per cent of those which showed marked changes (2 per cent of the total group). An acute suppurative process was present in 6, an acute and acute catarrhal process in 12 and a subacute process in 15. The subacute process lies between acute and chronic exudative appendicitis.

The records of the patients from whom the foregoing 33 appendixes were removed were rechecked for any history or physical findings which would even suggest the possibility of appendical disease at the time of the operation or previously. None could be found. Three of the acute suppurative appendixes were removed from my own personal patients. In each instance, after the pathologic changes were reported the history was rechecked with the patient. No overlooked points in the history which pointed in any way to the presence of appendicitis were disclosed.

One must bear in mind the possibility that the pathologic changes present in some of the appendixes which presented acute or subacute, but not suppurative, inflammation might have been produced by the appendix having been packed away tightly with pads while a long primary operation was being performed.

Chronic obliterative appendicitis with infiltration likewise requires no definition, as the name gives a complete description of the pathologic lesion. The mucosa and often the other coats, even including a part of the muscle, have disappeared and been replaced by granulations or scar tissue, which, with the muscle, shows a definite infiltration. The process may involve a part of or the whole appendix. It probably represents either a stage in the healing of a suppurative or ulcerative process or a renewed inflammation in an appendix which had previously healed. This condition was observed in 106 appendixes, or 17 per cent of those showing marked changes (6 per cent of the whole group).

Chronic obliterative appendicitis without infiltration is, as the name designates, the same as the foregoing condition with the exception that it is a completely healed process without inflammation. One hundred and eighty-three appendixes were so effected, or 30 per cent of those showing marked pathologic changes (10 per cent of the total group). Of the 7 groups of appendixes which presented marked inflammatory changes, this was the largest.

Chronic periappendicitis is associated with and due to a chronic inflammatory lesion elsewhere in the abdomen, usually chronic salpingitis. This pathologic condition was present in 31 appendixes, or only 5 per cent of those showing marked changes (2 per cent of the total group). The number was surprisingly small considering the large number of cases of salpingitis included in the list.

Acute periappendicitis constitutes its own definition. This condition is associated with and due to an acute inflammatory lesion elsewhere in the abdomen. It was found in only 23 appendixes, or 3.7 per cent of those which showed marked changes (1 per cent of the total group). This group was small because ordinarily the primary lesion in the abdomen and the patient's condition were contraindications to doing an incidental appendectomy.

MORTALITY ACCORDING TO CONDITION FOUND IN THE APPENDIX

The mortality figures are given only for completeness. The pathologic condition found in these appendixes had no bearing on any of the

TABLE 2.—*Classification of Pathologic Conditions of Appendixes, With the Incidence and Mortality for Each Group*

Condition in Appendix	Number of Appendixes	Percentage*	Number of Deaths	Mortality, Percentage
No microscopic examination	161	....	1	0.62
Mucocele	3	0.16	0	0.00
Tuberculosis	5	0.26	1	20.00
Tumor	6	0.32	0	0.00
No inflammation	702	36.9	5	0.71
Normal 629		33.1	4	0.62
Atrophic 73		3.8	1	1.37
Inflammation	1,188	62.4	20	1.67
Simple chronic appendicitis 573		30.1	10	1.75
Marked inflammatory changes 615		32.3	10	1.63
Chronic catarrhal appendicitis 82		4.3	1	1.22
Chronic exudative appendicitis 157		8.3	2	1.27
Acute appendicitis 33		1.7	0	0.00
Chronic obliterative appendicitis with infiltration 106		5.6	1	0.94
Chronic obliterative appendicitis without infiltration 183		9.6	4	2.19
Chronic periappendicitis 31		1.6	0	0.00
Acute periappendicitis 23		1.2	2	8.70
Totals	2,065	100.00	27	1.31

* The percentages are calculated only on the 1,904 appendixes examined microscopically.

deaths in this list. Any increase above the average is due to the primary operation or to the indication for that operation.

The number of tuberculous appendixes was too small for the mortality percentage to be significant. The single death was due to generalized tuberculosis.

Chronic obliterative appendicitis with infiltration is found to a large extent in older patients. They are poorer operative risks and more often require extensive primary operations for more serious conditions than younger patients. Possibly this argues that they should not undergo an incidental appendectomy, although the mortality for the group reported here is no greater than would be expected had the appendectomy not been done.

The increase in mortality in the group of patients with acute periappendicitis was caused by the death of 2 with advanced generalized

peritonitis due to a perforated diverticulum of the sigmoid colon. Undoubtedly these patients should not have been subjected to an appendectomy.

RELATION OF INFLAMMATORY CHANGE IN THE APPENDIX TO INFLAMMATION ELSEWHERE IN THE ABDOMEN

The relationship of inflammatory change in the appendix to inflammation elsewhere in the abdomen, without taking into consideration the type or location of the latter, is shown in table 3. This and all subsequent percentage charts have been so arranged that comparisons may be made by reading down the columns of percentages. Any effect that

TABLE 3.—*Relation of the Inflammatory Change in the Appendix to the Incidence of Inflammation Elsewhere in the Abdomen* *

Condition in Appendix	Total Appendixes	Condition Elsewhere in Abdomen: No Inflammation Number	No Inflammation Percentage	Inflammation Number	Inflammation Percentage
No inflammation	702	465	66.2	237	33.8
Normal	629	415	66.0	214	34.0
Atrophic	73	50	68.5	23	31.5
Inflammation	1,188	674	56.8	514	43.2
Simple chronic appendicitis	573	320	55.8	253	44.2
Marked inflammatory changes	570	354	62.1	216	37.9
Chronic catarrhal appendicitis	82	55	67.0	27	33.0
Chronic exudative appendicitis	157	94	59.8	63	40.2
Acute appendicitis	33	23	69.7	10	30.3
Chronic obliterative appendicitis with infiltration	106	59	55.7	47	44.3
Chronic obliterative appendicitis without infiltration	183	119	65.0	64	35.0
Chronic periappendicitis	31	4	12.9	27	87.1
Acute periappendicitis	23	0	00.0	23	100.0
Totals	1,890	1,139	60.3	751	39.7

* For comparisons read the vertical percentage columns. This table is elaborated in tables 4 and 5.

the items listed on the left had on the items listed at the top of the table are shown by variations in the vertical columns of figures.

The distribution of the appendixes according to the various types and locations of inflammation elsewhere in the abdomen is presented in table 4, and the corresponding incidences, figured on the total number of appendixes listed under each type of pathologic condition found elsewhere in the abdomen, in table 5. A slight increase in the incidence of appendixes showing inflammatory change accompanied the presence of inflammation elsewhere in the abdomen. This increase was greatest in the groups of appendixes affected with periappendicitis. Here, of course, the cause of this increase was elsewhere than in the appendix, and the relation is self-evident.

No cases of acute appendicitis were included under the headings chronic suppurative and acute salpingitis. In looking up the records, 30

TABLE 4.—*Distribution of Appendixes According to the Various Types of Inflammation Occurring Simultaneously Elsewhere in the Abdomen*

Condition in Appendix	Condition Elsewhere in Abdomen											
	No Inflammation	Inflammation	Salpingitis	Chronic Salpingitis	Chronic Suppurative Salpingitis	Acute Salpingitis	Cholecystitis	Peptic Ulcer	Tuberculous Peritonitis or Salpingitis	Peritonitis	Other Types of Inflammation	Total Appendixes
No inflammation	465	237	145	125	9	11	74	13	3	1	1	702
Normal	415	214	135	115	9	11	63	12	2	1	1	629
Atrophic	50	23	10	10	0	0	11	1	1	0	0	73
Inflammation	674	514	306	202	50	54	161	29	5	6	7	1,188
Simple chronic appendicitis	320	253	148	105	30	13	80	13	5	3	4	573
Marked inflammatory changes	354	261	158	97	20	41	81	16	0	3	3	615
Chronic catarrhal appendicitis	55	27	20	16	1	3	7	0	0	0	0	82
Chronic exudative appendicitis	94	63	39	27	2	10	18	5	0	0	1	157
Acute appendicitis	23	10	2	2	0	0	7	0	0	1	0	33
Chronic obliterative appendicitis with infiltration	59	47	19	13	2	4	19	7	0	0	2	106
Chronic obliterative appendicitis without infiltration	119	64	30	25	2	3	30	4	0	0	0	183
Chronic periappendicitis	4	27	27	13	13	1	0	0	0	0	0	31
Acute periappendicitis	0	23	21	1	0	20	0	0	0	2	0	23
Totals	1,139	751	451	327	59	65	235	42	8	7	8	1,890

TABLE 5.—*Distribution of Appendixes According to Various Types of Inflammation Occurring Simultaneously Elsewhere in the Abdomen (Percentages)* *

Condition in Appendix	Condition Elsewhere in Abdomen											
	Total Appendixes	No Inflammation	Inflammation	Salpingitis	Chronic Salpingitis	Chronic Suppurative Salpingitis	Acute Salpingitis	Cholecystitis	Peptic Ulcer	Tuberculous Peritonitis or Salpingitis	Peritonitis	Other Types of Inflammation
No inflammation	702	66.24	33.76	20.65	17.81	1.28	1.56	10.45	1.85	0.44	0.14	0.14
Normal	629	65.97	34.03	21.46	18.28	1.43	1.75	10.02	1.91	0.32	0.16	0.16
Atrophic	73	68.49	31.51	13.69	13.69	0.00	0.00	15.07	1.37	1.37	0.00	0.00
Inflammation	1,188	56.73	43.27	25.76	17.00	4.21	4.55	13.55	2.44	0.43	0.50	0.59
Simple chronic appendicitis	573	55.85	44.15	25.83	18.32	5.23	2.27	13.96	2.27	0.87	0.52	0.70
Marked inflammatory changes	615	57.56	42.44	25.76	15.77	3.25	6.67	13.17	2.60	0.00	0.49	0.49
Chronic catarrhal appendicitis	82	67.07	32.93	24.39	19.51	1.22	3.66	8.54	0.00	0.00	0.00	0.00
Chronic exudative appendicitis	157	59.87	40.13	24.83	17.19	1.27	6.37	11.47	3.19	0.00	0.00	0.64
Acute appendicitis	33	69.70	30.30	6.06	6.06	0.00	0.00	21.21	0.00	0.00	3.03	0.00
Chronic obliterative appendicitis with infiltration	106	55.66	44.34	17.92	12.26	1.89	3.77	17.92	6.61	0.00	0.00	1.89
Chronic obliterative appendicitis without infiltration	183	65.03	34.97	16.39	13.66	1.09	1.64	16.39	2.19	0.00	0.00	0.00
Chronic periappendicitis	31	12.90	87.09	87.09	41.93	41.93	3.23	0.00	0.00	0.00	0.00	0.00
Acute periappendicitis	23	0.00	100.00	91.30	4.35	0.00	86.96	0.00	0.00	0.00	8.70	0.00
Figures for whole group	1,890	60.26	39.74	23.86	17.30	3.12	3.44	12.43	2.22	0.43	0.37	0.43

* The percentage incidence was calculated on the total number of appendixes classified with each type of pathologic condition found elsewhere in the abdomen. For comparisons, read the vertical columns.

cases were found in which subacute, acute or acute suppurative appendicitis occurred simultaneously with one of these two conditions when the indication for the operation was a pathologic condition of the tubes. However, they were all discarded as it was impossible to state positively that the symptoms were not at least partially due to the inflammatory process in the appendix.

One portion of this table is interesting because of the definite lack of relationship shown and will be taken up in detail later.

RELATION OF INFLAMMATORY CHANGE IN THE APPENDIX TO INFLAMMATION IN THE GALLBLADDER

The statement has often been made that inflammation in the gallbladder is largely, or at least frequently, caused by a chronic inflam-

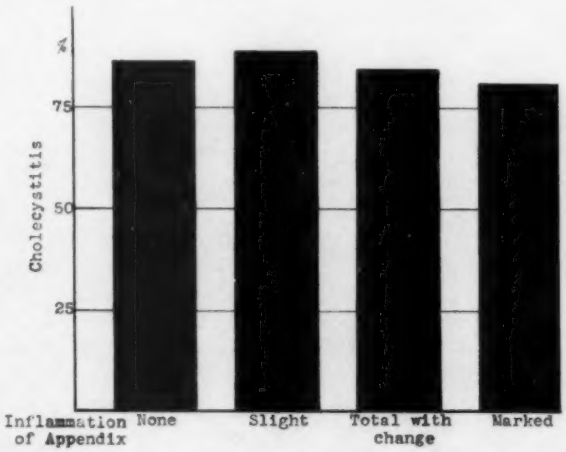

Chart 1.—Relation of inflammatory change in the appendix to the incidence of inflammation in the gallbladder. The incidence of the latter does not increase in the presence of inflammatory change in the appendix.

matory process in the appendix. Tables 6 and 7 and charts 1 and 2 present the statistics for cases in which the gallbladder and appendix were removed simultaneously. If inflammation in the appendix causes inflammation in the gallbladder, the incidence of cholecystitis should show a definite increase when inflammation is found in the appendix, and one might expect that increase to be greater the more marked the inflammatory change in the appendix. The tables and charts do not show this. In fact, there is a slight variation in the opposite direction (table 6 and chart 1). Conversely, the incidence of normal gallbladders should be decreased in the presence of inflammatory change in the appendix. As before, a slight variation in the opposite direction is found (table 6 and chart 2).

The same relation is presented in table 7, but the incidence of inflammatory change in the appendix was calculated on the number of normal gallbladders and those showing cholecystitis. The same lack of causative relationship is seen.

A careful examination of these figures discloses no evidence to substantiate the statement that chronic inflammation in the appendix causes inflammation in the gallbladder.

TABLE 6.—*Relation of Inflammatory Change in the Appendix to Inflammation in the Gallbladder* *

Condition in Appendix	Total Appendixes	Condition in Gallbladder: Normal — Number	Normal — Percentage	Cholecystitis — Number	Cholecystitis — Percentage
No inflammation	86	12	13.9	74	86.1
Inflammation	190	29	15.3	161	84.7
Slight	90	10	11.1	80	88.9
Marked	100	19	19.0	81	81.0
Totals	276	41	14.8	235	85.2

* This table indicates that inflammation in the appendix is not a cause of inflammation in the gallbladder. If it were, an increase in the incidence of cholecystitis should be found in the presence of inflammatory change in the appendix. There is instead a slight decrease in incidence. This table is shown graphically in charts 1 and 2.

TABLE 7.—*Relation of Inflammation in the Gallbladder to Inflammatory Change in the Appendix* *

Condition in Gallbladder	Total Cases	Inflammatory Change in Appendix: None — Number	None — Percentage	Total — Number	Total — Percentage	Slight — Number	Slight — Percentage	Marked — Number	Marked — Percentage
Normal	41	12	29.1	29	70.9	10	24.4	19	46.5
Cholecystitis	235	74	31.5	161	68.5	80	34.2	81	34.3
Totals	276	86	31.1	190	68.9	90	32.6	100	36.4

* This table indicates that in the presence of inflammation in the gallbladder there is no increase in the incidence of inflammatory change in the appendix.

RELATION OF INFLAMMATION ELSEWHERE IN THE ABDOMEN TO INFLAMMATORY CHANGE IN THE APPENDIX

The distribution of inflammatory conditions in the abdomen and their proportionate frequency, without taking into consideration the type of inflammatory change in the appendix, is presented in table 8. The distribution is shown in detail in table 9, and the corresponding incidences, calculated on the total number of cases in each group, in table 10.

By reading down the percentage columns in table 10 under each type of pathologic process found in the appendix, the effect, if any, on each pathologic process of inflammation elsewhere in the abdomen can be readily seen. An extensive discussion is unnecessary.

In the presence of inflammation elsewhere in the abdomen there was a definite increase in the incidence of inflammatory change in the appendix. This same relation held true with simple chronic appendicitis, marked inflammatory changes (total), chronic obliterative appendicitis with infiltration and, as one would expect, to a much greater extent with chronic and acute periappendicitis. No causative relationship was indicated in chronic catarrhal appendicitis, chronic exudative appendicitis, acute appendicitis and chronic obliterative appendicitis without infiltration. Apparently inflammation elsewhere in the abdomen has no appreciable effect on any of the latter group of changes found in the appendix. The only really outstanding relationship shown was that of salpingitis and peritonitis in causing acute and chronic periappendicitis.

What appears to be an abnormal variation under chronic obliterative appendicitis (both groups) was due to what is later shown to be age incidence. Here the higher percentages were found in those conditions

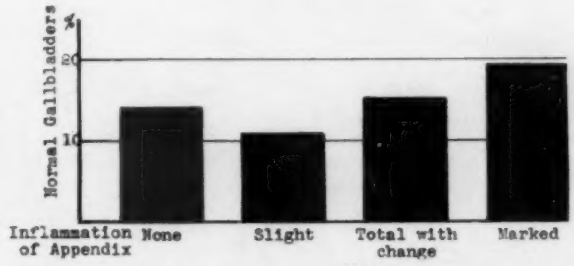

Chart 2.—Relation of inflammatory change in the appendix to the incidence of normal gallbladders. The incidence of the latter does not decrease in the presence of inflammatory change in the appendix.

occurring chiefly in middle and later life and the low percentages in conditions found in the early decades of life. This same relationship can be seen to a lesser degree in other parts of this table.

INCIDENCE OF INFLAMMATORY CHANGE IN THE APPENDIX ACCORDING TO AGE

In discussing the relationship of inflammatory change in the appendix to age, the age groups from 1 to 10 and from 71 to 80 will be ignored because of the small number of patients in each. The total number in groups from 11 to 20 and from 61 to 70 were much smaller than in those remaining, but, since there are sufficient to figure percentages and in each the resultant figures fell well within the expected percentages, they have been used without weighting.

The incidence of the total number of appendixes showing inflammatory changes increased definitely with age. However, the incidence

of those showing the various types of inflammation varied considerably from that in the total group. These differences are shown graphically in charts 3 to 16.

The increase of incidence of the appendixes with inflammatory change which occurred with an increase in age and the corresponding decrease in the number of appendixes without inflammatory change can be seen in chart 3 (solid lines). A more uniform decrease with age through the middle decades was found by eliminating the atrophic appendixes and considering only the normal appendixes. When the atrophic appendixes were added to those showing inflammatory change,

TABLE 8.—*Effect of Inflammation Elsewhere in the Abdomen on the Incidence of Inflammation in the Appendix* *

Condition Elsewhere in Abdomen	Total Cases	Condition in Appendix: No Inflammation		Condition in Appendix: Inflammatory Change	
		Number	Percentage	Number	Percentage
No inflammation	1,139	465	40.8	674	59.2
Inflammation	751	237	31.6	514	68.4
Salpingitis	451	147	32.1	306	67.9
Chronic salpingitis	327	125	38.2	202	61.8
Chronic supurative salpingitis	59	9	15.2	50	84.8
Acute salpingitis	65	11	18.5	54	81.5
Cholecystitis	235	74	31.5	161	68.5
Peptic ulcer	42	13	30.9	29	69.1
Tuberculous peritonitis or salpingitis	8	3	37.5	5	62.5
Peritonitis	7	1	14.3	6	85.7
Other types of inflammation	8	1	12.5	7	87.5
Totals	1,890	702	37.1	1,188	62.9

* For comparisons, read the vertical percentage columns. Apparently the incidence of inflammation in the appendix is increased slightly by the presence of inflammation elsewhere in the abdomen. This table is elaborated in tables 9 and 10.

a correspondingly more uniform increase of the changes in the appendix with age was obtained (broken lines). This is a better representation of the effect of age on the appendix.

The incidence of the total number of appendixes without inflammation decreased gradually with age (chart 4). The decrease in the incidence of normal appendixes was slightly more marked (chart 5). The increase in the incidence of atrophic appendixes with an increase in age is represented in chart 6.

The incidence of the total number of appendixes showing inflammatory change increased with age (chart 7). However, there was a fairly stationary period during middle life. The trend or complete lack of trend is much more definitely seen in the various groups which make up this total.

TABLE 9.—*Distribution of the Various Types of Inflammation Found in the Appendix According to the Type of Inflammation Found Elsewhere in the Abdomen*

Condition Elsewhere in Abdomen	Condition in Appendix													
										Chronic Obliterative Appendicitis				
	No Inflammation	Normal	Atrophic	Inflammation	Simple Chronic Appendicitis	Marked Inflammatory Changes	Chronic Catarrhal Appendicitis	Chronic Exudative Appendicitis	Acute Appendicitis	With Infiltration	Without Infiltration	Chronic Periappendicitis	Acute Periappendicitis	Total Cases
No inflammation	465	415	50	674	320	354	55	94	23	59	119	4	0	1,139
Inflammation	237	214	23	514	253	261	27	63	10	47	64	27	23	751
Salpingitis	145	135	10	306	148	158	20	39	2	19	30	27	21	451
Chronic salpingitis	125	115	10	202	105	97	16	27	2	13	25	13	1	327
Chronic suppurative salpingitis	9	9	0	50	30	20	1	2	0	2	2	13	0	59
Acute salpingitis	11	11	0	54	13	41	3	10	0	4	3	1	20	65
Cholecystitis	74	63	11	161	80	81	7	18	7	19	30	0	0	235
Peptic ulcer	13	12	1	29	13	16	0	5	0	7	4	0	0	42
Tuberculous peritonitis or salpingitis	3	2	1	5	5	0	0	0	0	0	0	0	0	8
Peritonitis	1	1	0	6	3	3	0	0	1	0	0	0	2	7
Other types of inflammation	1	1	0	7	4	3	0	1	0	2	0	0	0	8
Total appendixes	702	629	73	1,188	573	615	82	157	33	106	183	31	23	1,890

TABLE 10.—*Distribution of the Various Types of Inflammation Found in the Appendix According to the Type of Inflammation Found Elsewhere in the Abdomen (Percentages)* *

Condition Elsewhere in Abdomen	Condition in Appendix													
											Chronic Obliterative Appendicitis			
	Total Cases	No Inflammation	Normal	Atrophic	Inflammation	Simple Chronic Appendicitis	Marked Inflammatory Changes	Chronic Catarrhal Appendicitis	Chronic Exudative Appendicitis	Acute Appendicitis	With Infiltration	Without Infiltration	Chronic Peri-appendicitis	Acute Peri-appendicitis
No inflammation	1,139	40.82	36.52	4.30	59.18	28.10	31.10	4.83	8.25	2.02	5.18	10.45	0.35	0.00
Inflammation	751	31.56	28.50	3.06	68.44	33.69	34.75	3.59	8.39	1.33	6.26	8.52	3.59	3.06
Salpingitis	451	32.15	29.94	2.21	67.85	32.82	35.03	4.21	8.63	0.44	4.21	6.65	5.99	4.65
Chronic salpingitis	327	38.22	35.32	2.90	61.78	32.12	29.66	4.90	8.30	0.62	3.98	7.65	3.98	0.32
Chronic suppurative salpingitis	59	15.25	15.25	0.00	84.75	50.85	33.90	1.69	3.39	0.00	3.39	3.39	22.03	0.00
Acute salpingitis	65	18.46	18.46	0.00	81.54	18.46	63.08	4.62	14.62	0.00	6.15	4.62	1.54	29.24
Cholecystitis	235	31.49	26.81	4.68	68.51	34.04	34.47	2.98	7.67	2.98	8.09	12.77	0.00	0.00
Peptic ulcer	42	30.95	28.27	2.38	69.05	30.96	38.09	0.00	11.90	0.00	16.67	9.52	0.00	0.00
Tuberculous peritonitis or salpingitis	8	37.50	25.00	12.50	62.50	62.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Peritonitis	7	14.29	14.29	0.00	85.71	42.85	42.85	0.00	0.00	14.29	0.00	0.00	0.00	28.58
Other types of inflammation	8	12.50	12.50	0.00	87.50	50.00	37.50	0.00	12.50	0.00	25.00	0.00	0.00	0.00
Figures for whole group	1,890	37.15	33.28	3.87	62.85	30.31	32.54	4.34	8.31	1.75	5.61	9.68	1.64	1.22

* The percentage incidence was calculated on the total number of appendixes classified with each type of inflammation elsewhere in the abdomen. For comparisons, read the vertical columns.

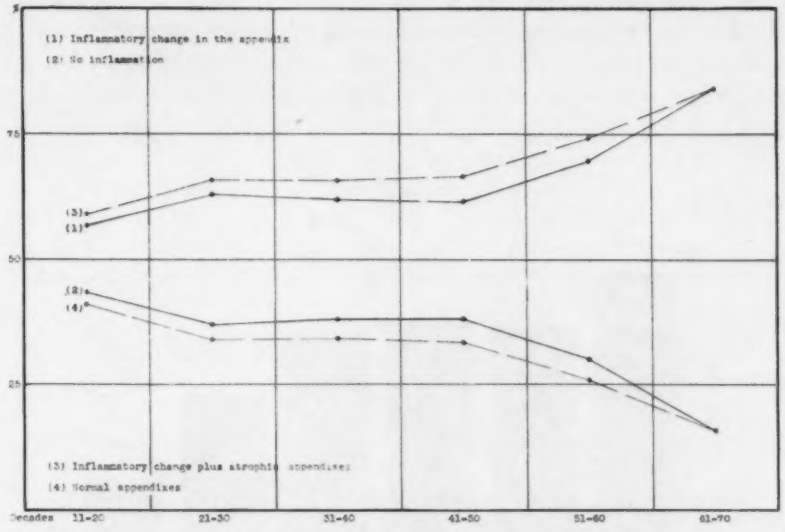

Chart 3.—The effect of age on the incidence of changes in the appendix. The comparison is shown of no inflammation to inflammatory change (solid lines) and also of normal appendixes to those showing atrophic and inflammatory change (broken lines).

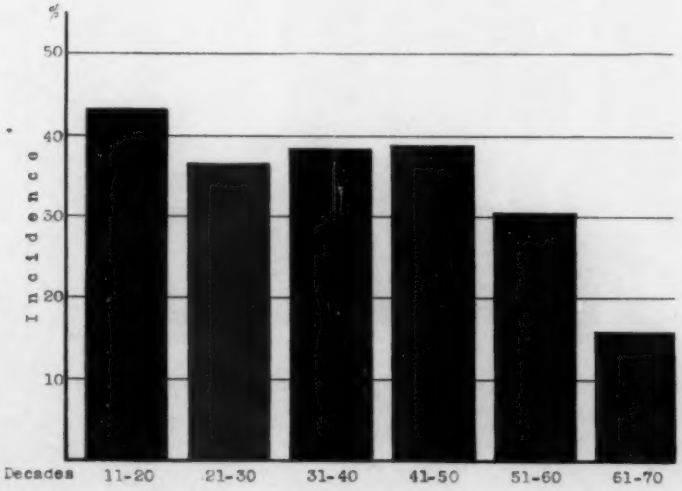

Chart 4.—Incidence of appendixes which presented no inflammatory change according to age.

There was a slight but definite decrease in the incidence of simple chronic appendicitis with age (chart 8). This might be accounted for by the fact that appendixes with more marked pathologic processes as age increases come from this group as well as from the group of normal appendixes. The total number of appendixes with marked changes

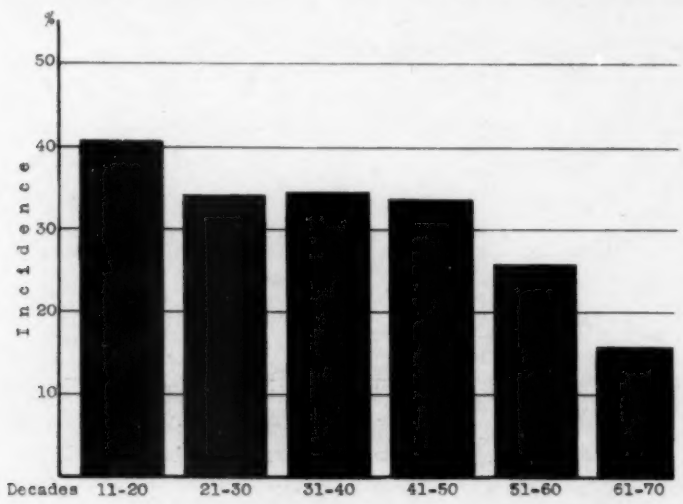

Chart 5.—Incidence of normal appendixes according to age. Those appendixes which were relatively normal, i. e., showed no definite inflammatory change, were also included.

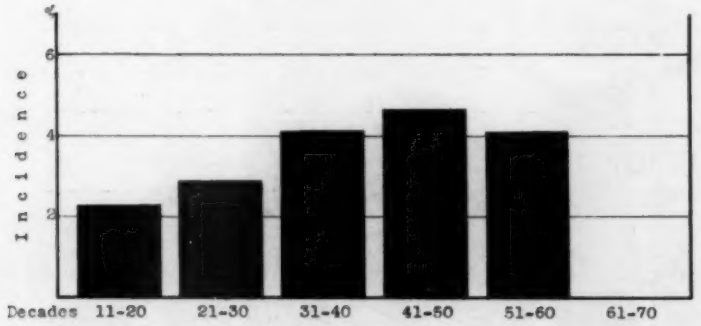

Chart 6.—Incidence of atrophic appendixes according to age.

increased in incidence with age (chart 9). There was a slight increase in the incidence of chronic catarrhal appendicitis with an increase in age (chart 10). There was a marked increase with age in the incidence of chronic obliterative appendicitis, both with (chart 11) and without infiltration (chart 12). The incidence of chronic exudative appendicitis (chart 13) and acute appendicitis (chart 14) showed a spread

throughout, indicating that age had no effect. This is of particular interest as pathologically these conditions are closely related.

There was an increase in the incidence of periappendicitis, both chronic (chart 15) and acute (chart 16), in the early age group and a marked decrease with age. This is exactly as would be expected, because, as mentioned earlier, the causes of periappendicitis are largely conditions occurring in early and early middle life.

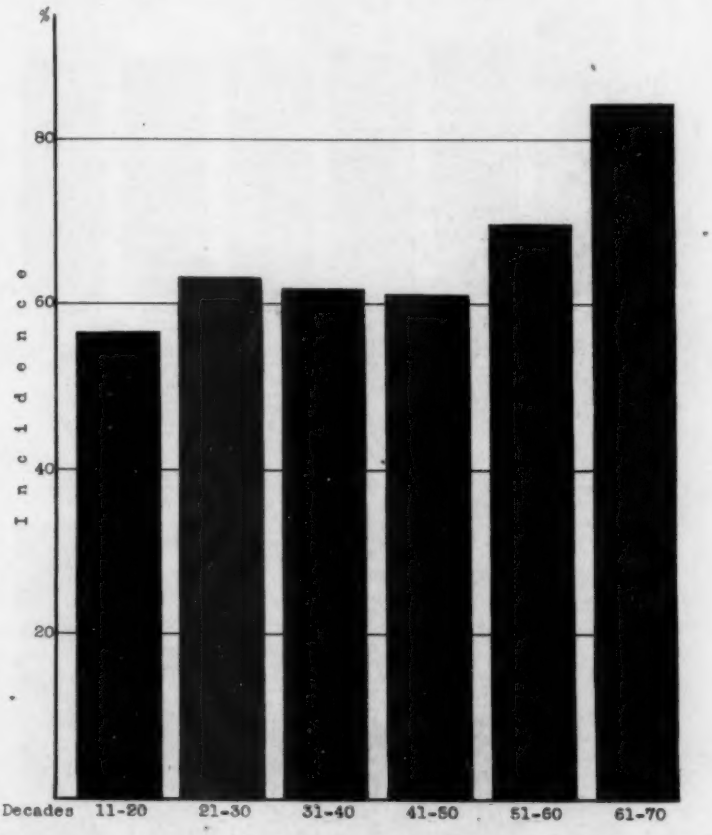

Chart 7.—Incidence of total appendixes with inflammatory change according to age.

To summarize the effect of age on the incidence of inflammatory changes found in these appendixes:

There was an increased incidence of inflammatory change with increasing age of the patients from whom the appendixes were removed (charts 3 and 7). This increase in pathologic change was found slightly greater when the appendixes which showed atrophic changes were added to those showing inflammatory change (chart 3).

The incidence of appendixes without inflammatory change (charts 3 and 4) and of the appendixes with chronic periappendicitis (chart 15) and acute periappendicitis (chart 16) decreased with increasing age.

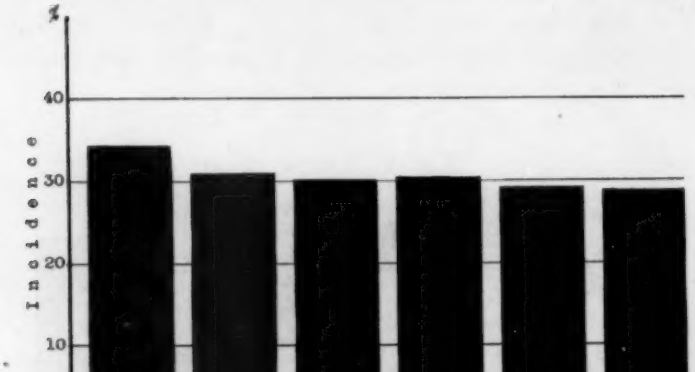

Chart 8.—Incidence of simple chronic appendicitis according to age.

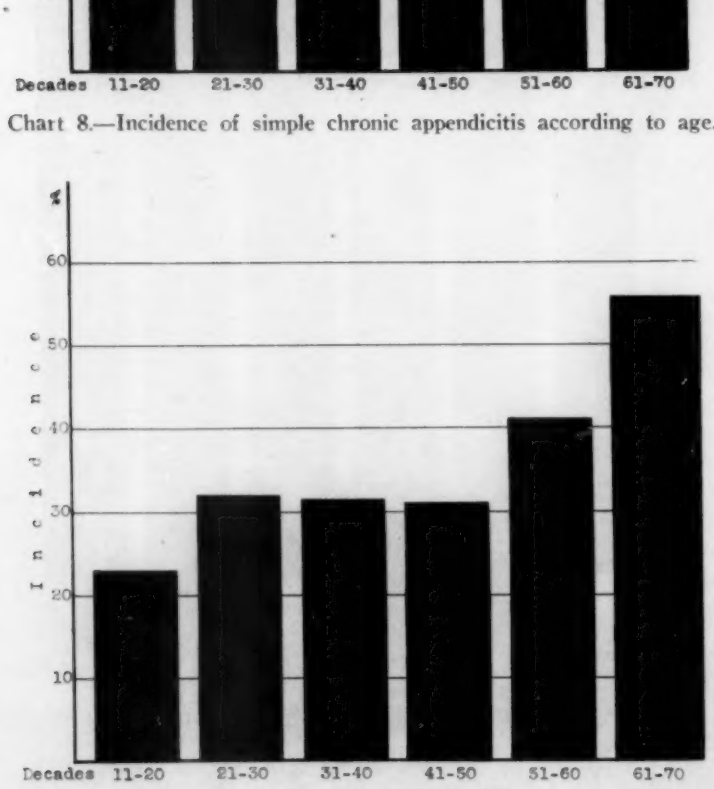

Chart 9.—Incidence of appendixes with marked inflammatory change according to age.

The incidence of chronic exudative appendicitis (chart 9) and acute appendicitis (chart 10) showed no increase with an increase in age.

TABLE 11.—*Incidence of Inflammatory Change in the Appendix According to Age*

Age by Decades	Condition in Appendix													
										Chronic Obliterative Appendicitis				
	No Inflammation	Normal	Atrophic	Inflammation	Simple Chronic Appendicitis	Marked Inflammatory Changes	Chronic Catarrhal Appendicitis	Chronic Exudative Appendicitis	Acute Appendicitis	With Infiltration	Without Infiltration	Chronic Periappendicitis	Acute Periappendicitis	Total Cases
1 to 10	1	1	0	4	2	2	0	0	1	0	0	0	1	5
11 to 20	19	18	1	25	15	10	0	3	1	0	1	1	4	44
21 to 30	151	139	12	258	127	131	20	38	3	14	27	16	13	409
31 to 40	274	245	29	441	215	226	41	56	17	34	63	12	3	715
41 to 50	207	182	25	331	163	168	10	40	8	39	68	2	1	538
51 to 60	46	40	6	106	43	63	9	18	3	13	19	0	1	152
61 to 70	4	4	0	21	7	14	2	2	0	6	4	0	0	25
71 to 80	0	0	0	2	1	1	0	0	0	0	1	0	0	2
Totals	702	629	73	1,188	573	615	82	157	33	106	183	31	23	1,890

TABLE 12.—*Percentage Incidence of Various Types of Inflammatory Change in the Appendix According to Age* *

Age by Decades	Total Cases	Condition in Appendix												
											Chronic Obliterative Appendicitis			
		No Inflammation	Normal	Atrophic	Inflammation	Simple Chronic Appendicitis	Marked Inflammatory Changes	Chronic Catarrhal Appendicitis	Chronic Exudative Appendicitis	Acute Appendicitis	With Infiltration	Without Infiltration	Chronic Periappendicitis	Acute Periappendicitis
1 to 10	5	20.00	20.00	0.00	80.00	40.00	40.00	0.00	0.00	20.00	0.00	0.00	0.00	20.00
11 to 20	44	43.18	40.91	2.27	56.82	34.09	22.73	0.00	6.82	2.28	0.00	2.28	2.28	9.07
21 to 30	409	36.82	33.98	2.84	63.18	31.05	32.13	4.89	9.29	0.74	3.42	6.64	3.91	3.18
31 to 40	715	38.32	34.23	4.00	61.68	30.07	31.61	5.73	7.83	2.38	4.76	8.81	1.68	0.42
41 to 50	538	38.47	33.83	4.64	61.53	30.29	31.24	1.85	7.44	1.49	7.25	12.64	0.37	0.18
51 to 60	152	30.26	26.18	4.08	69.74	28.29	41.45	5.92	11.84	1.97	8.56	12.50	0.00	0.66
61 to 70	25	16.00	16.00	0.00	84.00	28.00	56.00	8.00	8.00	0.00	24.00	16.00	0.00	0.00
71 to 80	2	0.00	0.00	0.00	100.00	50.00	0.00	0.00	0.00	0.00	0.00	50.00	0.00	0.00
Totals	1,890	37.15	33.28	3.87	62.85	30.31	32.54	4.34	8.31	1.75	5.61	9.68	1.64	1.22

* The percentages were calculated on the total number of appendixes in each decade. This table is shown graphically in charts 3 to 16.

The incidence of appendixes with inflammatory change (chart 7) and those with marked inflammatory change (chart 9) increased with age; there was also a similar increase in the incidence of chronic catar-

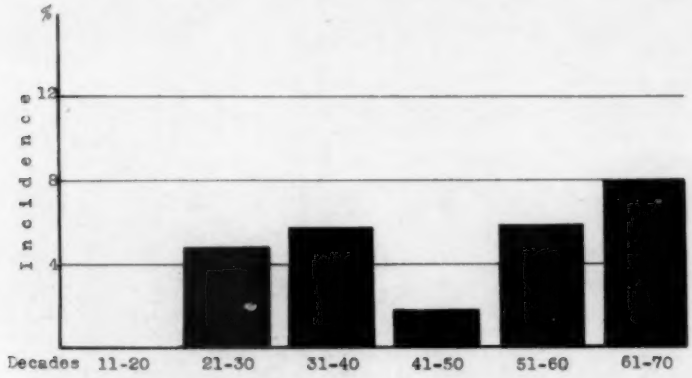

Chart 10.—Incidence of chronic catarrhal appendicitis according to age.

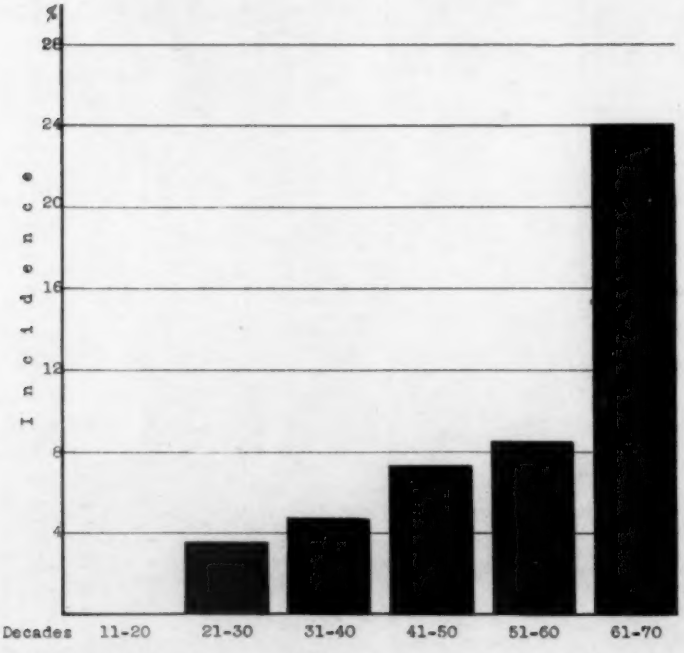

Chart 11.—Incidence of chronic obliterative appendicitis with infiltration according to age.

rhal appendicitis (chart 10), atrophic appendicitis (chart 6), chronic obliterative appendicitis without infiltration (chart 12) and chronic obliterative appendicitis with infiltration (chart 11).

OTHER PATHOLOGIC CONDITIONS

Oxyuris in the Lumen of the Appendix.—Three appendixes were found to contain Oxyuris in the lumen, an incidence of 0.2 per cent. The pathologic conditions in these appendixes were classified as follows:

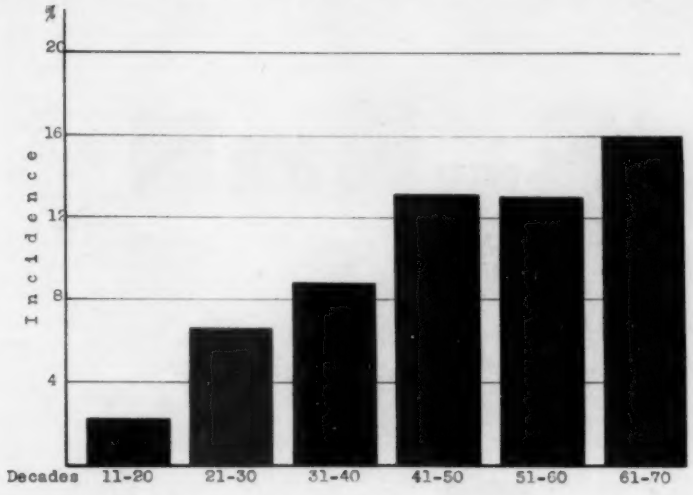

Chart 12.—Incidence of chronic obliterative appendicitis without infiltration according to age.

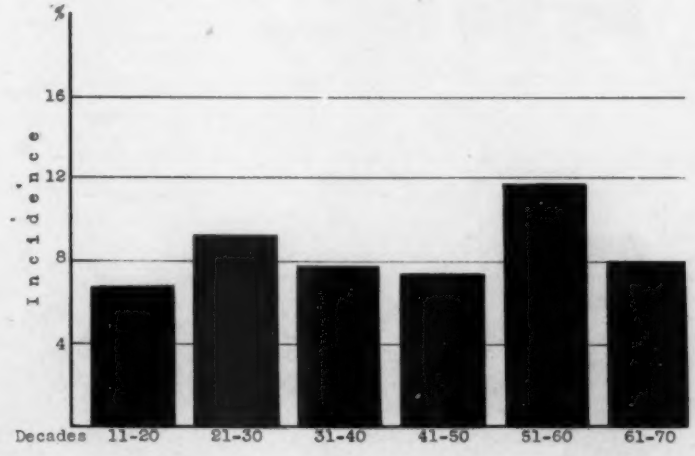

Chart 13.—Incidence of chronic exudative appendicitis according to age.

simple chronic appendicitis, chronic obliterative appendicitis with infiltration and acute catarrhal appendicitis. Two of the patients were operated on because of retroversion of the uterus and the other because of an ovarian cyst. Their ages were 30, 30 and 32 years.

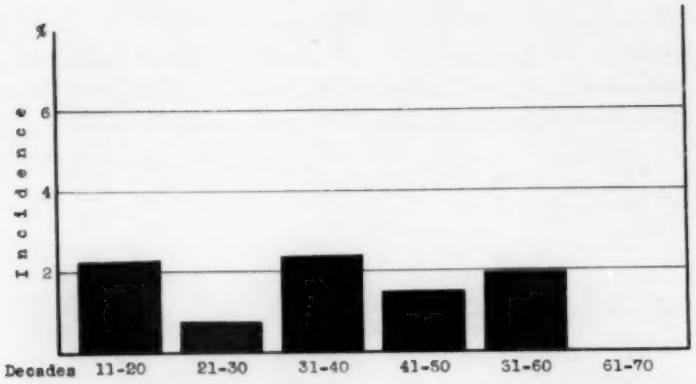

Chart 14.—Incidence of acute appendicitis according to age.

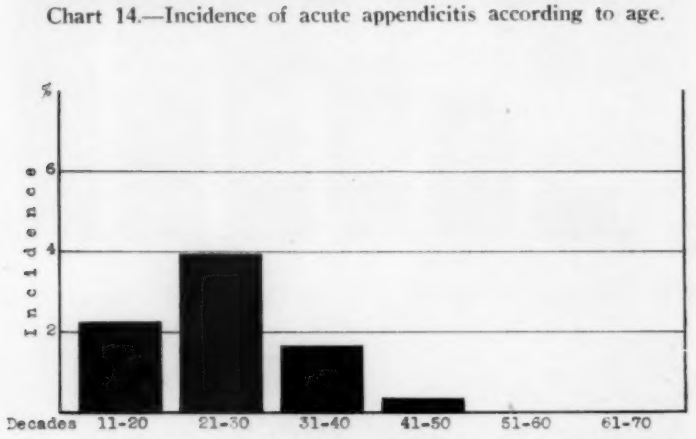

Chart 15.—Incidence of chronic periappendicitis according to age.

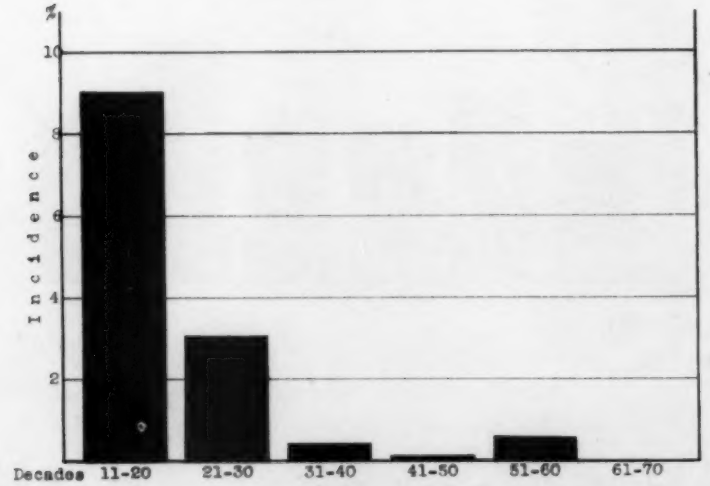

Chart 16.—Incidence of acute periappendicitis according to age.

Mucocele of the Appendix.—Mucoceles were found in 3 appendixes, an incidence of 0.2 per cent. One patient was operated on for intestinal obstruction due to adhesions and the other 2 for fibromyomata uteri. Their ages were 30, 44 and 75 years.

Tuberculosis of the Appendix.—This condition was found in 5 appendixes, an incidence of 0.3 per cent. Three appendixes showed chronic tuberculous appendicitis when no other tuberculous condition was noted in the abdomen. One was removed at the same time as the gallbladder, a diagnosis of chronic cholecystitis with cholelithiasis having been made. Another was removed at an operation for chronic salpingitis, but microscopic examination revealed no evidence of tuberculosis. The third was removed at the time of an operation for fibromyomata uteri.

TABLE 13.—*Pathologic Conditions Found in the Appendix, Other Than Inflammatory Change or Its Absence, With Incidence* *

Pathologic Condition	Number of Cases	Incidence, Percentage	Number of Deaths	Mortality, Percentage
Oxyuris in the lumen	3	0.16	0	0.00
Mucocele	3	0.16	0	0.00
Tuberculosis	5	0.26	1	20.00
Diverticulum	1	0.05	0	0.00
Hyperplasia of the mucosa	3	0.16	0	0.00
Tumors	6	0.32	0	0.00
Totals	21	1.11	1	4.76

* The incidence was calculated on the basis of the 1,904 appendixes examined microscopically.

I repeat for emphasis that the histories and physical examinations in these 3 cases and in the following cases revealed no symptom or sign referable to the appendix.

The fourth appendix presented chronic tuberculous appendicitis in conjunction with a tuberculous cecum, the original operation having been done for fibromyomata uteri and retroversion.

The fifth appendix was removed from a patient who had acute tuberculous peritonitis. Microscopically it showed subacute tuberculous appendicitis. The patient died of generalized tuberculosis.

The age incidence in these cases was 20, 32, 35, 38 and 42 years.

Syphilis.—Microscopic evidence of syphilis was not found throughout the series, although a number of the patients had positive Wassermann reactions.

Diverticulum of the Appendix.—One appendix contained a diverticulum, an incidence of 0.1 per cent. The original cause for operation was ectopic endometrium on the pelvic peritoneum. The patient was 34 years old.

Hyperplasia of the Mucosa.—Three appendixes showed marked hyperplasia of the mucosa, an incidence of 0.2 per cent; in 1 instance it

was described as papillary and in the other as adenomatous. This condition follows an inflammatory process and cannot be classified as a new growth in this stage. Two of the primary operations were performed for fibromyomata uteri and 1 for a parovarian cyst. The ages of these patients were 26, 38 and 42 years.

Tumors of the Appendix (table 14).—Four appendixes showed endometrial rests, an incidence of 0.2 per cent. Three were from patients operated on for fibromyomata uteri and the other from a patient operated on for retroversion and chronic salpingitis. Their ages were 38, 42, 44 and 50 years.

A carcinoid of the appendix (an incidence of 0.1 per cent) was found in a patient 45 years old who was operated on for fibromyomata uteri.

One appendix contained a papillary adenoma, possibly malignant (an incidence of 0.1 per cent). The patient, aged 26, was operated on because of a tubal pregnancy.

TABLE 14.—*Types of Tumors Found in the Appendixes Studied, With Their Incidence* *

Tumor	Number of Cases	Incidence, Percentage	Number of Deaths
Endometrial rests	4	0.21	0
Carcinoid	1	0.05	0
Papillary adenoma, possibly malignant	1	0.05	0

* This was calculated on the basis of the 1,904 examined microscopically.

It is of interest that none of the 1,904 appendixes examined microscopically showed a definite malignant process and that only 2 contained lesions which were questionably malignant.

SUMMARY AND CONCLUSIONS

Statistics on a group of 2,065 incidental appendectomies have been presented. These appendectomies were performed on patients whose histories and physical examinations gave no evidence of past or present appendical disease. The appendectomies included all those falling within the described definition which were done at St. Luke's Hospital (from both the private pavillion and the wards) in eleven years (1925 to 1935 inclusive). The pathologic conditions found in the 1,904 appendixes examined microscopically have been presented in the various tables and charts.

These findings may be stated briefly as follows: One third of the appendixes were either normal or relatively normal. Just less than one third showed slight to moderate, but definite, inflammation (simple chronic appendicitis). Over one third presented evidence of marked pathologic change. The latter group included those with types of appendicitis other than simple chronic appendicitis, together with atrophic

appendixes and those which showed other pathologic conditions, as listed in table 13.

Inflammatory changes are found in a considerable percentage of appendixes in the absence of a history or physical findings indicative of a pathologic condition in the right lower abdominal quadrant. In this series, 62 per cent fell within this class. More than half (51 per cent) of these showed marked inflammatory changes (32 per cent of the total number).

A slight but definite increase in inflammatory change in the appendix occurs in the presence of inflammation elsewhere in the abdomen. This becomes a marked increase with chronic suppurative and acute salpingitis and with peritonitis.

Acute appendicitis, even with actual suppuration, can exist without any subjective or objective evidence of its presence. The incidence was 1.7 per cent in this series.

A study of the cases in which the gallbladder was removed showed no increase in the incidence of cholecystitis in the presence of inflammation in the appendix. From this finding the statement can be made positively that no evidence is found to indicate that inflammation in the appendix causes inflammation in the gallbladder.

With increasing age there is a definite increase in the incidence of atrophic and inflammatory changes in the appendix. This is most marked with chronic obliterative appendicitis. It does not hold true for periappendicitis, which is caused by conditions prone to occur in early and early middle life.

The incidence of pathologic change other than inflammation is very low. This would indicate that these other conditions ordinarily produce symptoms referable to disease in the appendix.

When the appendix is not the cause of symptoms and there is no inflanimation elsewhere in the abdomen, the expected incidence of pathologic change in the appendix is represented by the top row of figures in table 10. I shall refer to these figures in a study of the symptom complex attributed to chronic appendicitis.

Although the figures are not presented, I feel that it is worth while to mention the fact that the incidence of postoperative complications was found to be no greater than would be expected in a similar list of operations in which appendectomy was not performed. Also, no increase in mortality was found. From this the conclusion may be drawn that in properly selected cases, i. e., in those in which the nature of the operation and the patient's condition are favorable, the incidental removal of the appendix is a safe and worth while procedure.

The pathologic examination of all the appendixes listed in this paper was made by Dr. Leila C. Knox, pathologist at St. Luke's Hospital.

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CHRONIC FUNCTIONAL LESIONS OF THE SHOULDER

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It is difficult to find a title sufficiently noncommittal and inclusive and yet specific enough to designate the things I wish to consider. The word chronic in the title is used in a literal sense and entirely without any implication of disease, although some of the bony changes associated with these lesions simulate some of those commonly attributed to arthritis. I believe that the lesions here considered are not primarily or to any considerable degree due to the extraordinary uses to which the upper extremities are put but are caused merely by the ordinary, often repeated, everyday uses. They are hour to hour, day to day, month to month, year to year and decade to decade effects. It must not be overlooked, however, that some of the structures affected by function may be weakened gradually and eventually rupture under relatively slight strains, evoking symptoms out of all proportion to the forces involved at the time.

In considering the production of the lesions in question, one must divest oneself of the idea that the shoulder joint is a marvel of mechanical perfection. Even a cursory survey of its architecture should correct such a mistaken view. One may, I presume, grant that the human shoulder may be adapted perfectly to the uses for which it may have been intended, but certainly no one can rightly maintain that it is perfectly adapted for the uses to which it is put. These uses require a truly universal joint, and the humeroscapular articulation is such a one only imperfectly and only by subjecting the surrounding soft parts to repeated trauma. Even slight trauma recurring in the same place for hours, days, weeks and months has a cumulative effect and may produce great changes. It is hence no wonder that the reparative powers of the structures concerned often are exceeded and that repeated slight trauma may provoke an obliterative endarteritis and sclerosis. Moreover, since the individual trauma may fall below the threshold of reflex protective responses, there may be little to forestall the cumulative effect, and it is important to remember that it is the repetition and not the range of motion that counts.

Although I shall confine my remarks to the shoulder, I do not mean to imply that the functional lesions under consideration here occur nowhere else in the body. They are commonest and most pronounced

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in the shoulder and also seem to occasion the greatest amount of discomfort and disability there. Some of them may occur anywhere else where motion is possible, no matter how slight the range, and they always become evident to the unaided eye after adequate repetition of the motion. However, I have seldom noticed them before the third decade of life. The average age of the bodies from which the specimens for the illustrations were taken was over 60 years.

LESIONS CONCERNED

The lesions vary greatly in their intensity and manifestations from joint to joint and person to person. Although abnormal or pathologic conditions may facilitate and also markedly accentuate them, I am deliberately excluding changes due to disease and am confining my attention to the effects of normal use alone. In the last analysis, of course, these lesions are traumatic, but they are not that in the customary meaning of that word. They undoubtedly are not accompanied by hemorrhage or some other phenomena associated with lesions ordinarily regarded as traumatic; yet the distinction is quantitative rather than qualitative. Among the lesions concerned are the following:

1. Fraying and partial destruction of the inner surface of the walls of the subdeltoid bursa and of the supra-acromial bursa, when present, and of the deeper bursae about the shoulder.

2. Fraying and partial or complete destruction of considerable portions of the articular capsule, both from within and from without, and of the coracohumeral and the inconstant superior and middle glenohumeral ligaments.

3. Fraying and partial or complete division or detachment of the tendon of the supraspinatus and partial destruction of that of the infraspinatus and of the long tendon of the biceps and that of the subscapularis.

4. Partial or complete erosion of the coraco-acromial ligament.

5. Perforation of the acromioclavicular articulation below and complete destruction of the deeper portion of the articular capsule.

6. Partial or complete dislocation of the pretubercular and the intertubercular portions of the long tendon of the biceps.

7. Thinning and complete local destruction of the articular cartilages, both hyaline and fibroid, in the humeroscapular and the acromioclavicular articulations.

8. Fraying of the under surface of the deltoid muscle and complete destruction of a large part of its middle portion.

9. Polishing and eburnation of the bony surfaces that come into movable contact after destruction of the overlying or intervening soft parts and cartilages.

10. Partial or total destruction of the articular fibrocartilage and of the articular disk and the cartilage on the clavicle and acromion; opening of the acromioclavicular joint from below with or without bony polishing, sometimes even in cases of ununited acromions.

11. Formation of a true coracoclavicular articulation with or without bony contact between the clavicle and the coracoid process and consequent polishing.

12. Polishing of the coracoid process from contact with the head and tuberosities of the humerus.

13. Considerable loss of bone from wear in the areas of bony contact.

CONDITION OF BURSAE

An anatomist seldom encounters smooth superficial synovial bursae in cadavers past middle age. Since the amount of fluid in a normal bursa is limited to mere moistening of its internal surface, normal bursae are not distended, and their walls hence are constantly in contact with each other except for a thin film of fluid. Were they lined by a layer of epithelium, endothelium or highly specialized mesothelium, such as that in the peritoneum, pleura and pericardium resting on a subserosa, one might expect the bounding membranes to become detached from the connective tissue on which they rest, but the absence of such a mesothelium and basement membrane and the existence of a gradual transition from the surface layer of flattened, stellate mesothelium to ordinary connective tissue make such a separation impossible. It follows from this that he who does a "synovectomy" really does not remove a membrane but merely destroys the innermost portion of the connective tissue of an articular capsule.

Instead of a smooth inner surface in synovial bursae, roughening and fraying and complete destruction of it are frequently encountered. Such conditions commonly exist in the subacromial and the supracapsular portion of the subdeltoid bursa and may be present in any or most of the thirteen or more bursae which may be associated with the humeroscapular articulation. It is mainly these portions of the surfaces of the subdeltoid bursa which are brought into firm contact with every abduction of the arm. Hence the rest of the roof and the floor of the bursa usually are intact and normal, even after the entire upper portion of the articular capsule has been destroyed and when bony polishing is present on the tuberosital portion of the humerus which comes into contact with the acromion. Whenever the subdeltoid bursa is large, it extends distally onto the shaft of the humerus beyond the tuberosities and under the coracoid process, but the walls of this portion may then also be wholly preserved. The areas of most intimate and frequent contact are always the first to show change, although it sometimes happens that one of three possible independent subcoracoid bursae may show complete destruction of its smooth inner surface, although the smooth surface of the two other contiguous bursae may be unaffected. It has often been asserted that roughening and destruction of the normal walls of bursae are the result of inflammation, or bursitis. While recognizing fully the changes that may be effected by chronic inflammation in bursae, the lesions here considered manifestly were not produced in that way, and it is well to emphasize that long-continued movement of soft parts on each other frequently effects roughening and fraying of the apposed structures. This, to be sure, applies also to articular cartilages, both hyaline and fibroid.

It is of great significance that similar effects of wear are met with also between structures which normally are not separated by bursae, such as the radius and the muscles and the tendons of the long abductor and the long and short extensor muscles of the thumb and the extensor carpi radialis longus muscle. Such an instance is represented in figure 1, a photograph of a specimen taken from a right forearm which showed no evidence of any pathologic condition or of injury, past or present. It will be recalled that loose connective tissue normally separates these structures in this region of the antibrachium. This tissue evidently was destroyed and the coapted surfaces of the muscles and tendons frayed by wear from often repeated rotation of the radius. This may have occurred under the pressure of a sling, a lever or a

Fig. 1.—Fraying on the surfaces of the tendons and bellies of the abductor pollicis longus, the flexor pollicis longus, the extensor pollicis brevis and the extensor carpi radialis longus and of the loose connective tissue and the underlying periosteum in the left arm of a man 44 years of age. The tendon of the brachioradialis was reflected distally, and the photograph was taken from the side and in front with the forearm in semipronation. The belly of the abductor pollicis longus is indicated by *1*, the deeper surface of the extensor carpi radialis longus and brevis by *2*, the belly of the flexor pollicis longus by *3*, the tendon of the flexor carpi radialis by *4*, the tendon of the brachioradialis, reflected, by *5* and the tendon of the palmaris longus, on the edge, by *6*. The arrow points to the most deeply worn portion of *3*.

snugly fitting arm band, although probably none of these things are indispensable factors.

CAPSULAR DEFECTS

The forces which destroy synovial surfaces must of necessity produce similar effects on the underlying or deeper, outer bounding structures. Hence the articular capsule of the shoulder and the associated tendons may become similarly affected. The superficial surface of the

portion of the capsule immediately proximal to the greater tuberosity usually is the one to suffer roughening and fraying first, although roughening may also be evident on the inner surface of the capsule where it comes into contact with the margin of the articular cartilage. Thinning of the capsule and of the tendon of the supraspinatus muscle likewise usually occurs first proximal to the greater tuberosity, and perforation of the tendon and capsule usually is first seen here, as shown in figure 2. It may, however, also occur more ventrally or anteriorly to the tendon of the supraspinatus muscle in

Fig. 2.—Fraying of the inner surface of the subdeltoid bursa and an oval defect in the distal portion of the supraspinatus tendon with its long diameter in a longitudinal direction, indicated by the arrow.

the region under the coracoid process and even in the dorsal portion of the capsule, which is associated with the tendon of the infraspinatus muscle, as shown in figures 3 and 4 and represented by Meyer [1] in

1. Meyer, A. W.: Further Observations upon Use-Destruction in Joints, J. Bone & Joint Surg. **4**:491-511, 1922. See also: Meyer, A. W.: Anatomical Specimens of Unusual Clinical Interest, Am. J. Orthop. Surg. **13**:86-95, 1915; Further Evidence of Attrition in the Human Body, Am. J. Anat. **34**:241-267, 1924; Spontaneous Dislocation of the Tendon of the Long Head of the Biceps Brachii: Report of Four Cases, Arch. Surg. **13**:109-119 (July) 1926; The Minuter Anatomy of Attrition Lesions, J. Bone & Joint Surg. **13**:341-360, 1931.

Fig. 3.—Right shoulder viewed from above, showing defects in the articular capsule anterior and posterior to the unruptured tendon of the supraspinatus. The dorsal defect is indicated by *1*, the long tendon of the biceps lying in a large ventral defect, by *2*, the coraco-acromial ligament by *3* and the clavicle by *4*.

Fig. 4.—Two large attrition defects (*1* and *2*) in the dorsal portion of the capsule of the shoulder. The tendons of the infraspinatus and teres minor were practically intact. The cartilaginous margin of the head of the humerus responsible for these defects can be seen clearly across the lower defect, forming a slightly irregular border below *2*. The two defects are separated by a narrow strip of capsule on either side of which the (white) articular cartilage of the head of the humerus can be seen.

figures 8 and 9. The latter defects always are produced from within but the former largely from without, and they may be present when all the other articular structures are practically normal. Whenever the margin of the acromion or its under surface is rough or studded with small periosteal osteophytes, the superficial layer of the capsule, the tendon of the supraspinatus muscle and the deeper, thicker portion of the capsule, which may all be detached from each other, forming separate layers, may show parallel longitudinal scoring or ribboning, as shown in figures 5 and 6.

In cases of maximal capsular destruction, the entire upper portion is absent, the tendon of the infraspinatus muscle is forced backward and downward and that of the subscapularis muscle may be detached completely from the lesser tuberosity, maintaining its attachment to the adjacent diaphysis only. However, one can always find the retracted, more proximal, portion of the capsule under the acromion, for although retracted and sometimes somewhat folded, it always presents a thin, sclerotic, falciform border.

It is of much moment in connection with the causation of capsular defects into the shoulder joint from above that they may occur on either side of an unruptured and intact tendon of the supraspinatus muscle. Sometimes they are present on both sides of this tendon and may be represented by mere longitudinal slits, such as those represented at *2* and *3* in figure *7* and in figure 8. The character and direction of these defects show that they could not have been produced by pull of the muscle or rupture of a tendon, least of all by rupture of that of the supraspinatus, and the entire absence of evidence of disease in these cases shows that it cannot have been a factor in their production.

Whenever the periosteum distal to the capsular and tendinous attachment on the greater tuberosity of the humerus and that under the acromion are destroyed through contact, polishing and eburnation of the underlying bone are inevitable if adequate motion continues. Since the periosteum possesses marked osteogenetic powers, small osteophytes or exostoses not infrequently develop in the areas undergoing denudation if the necessary conditions prevail. This is what one should expect, and whenever osseous roughening occurs on the distal margin or the under surface of the acromion, the destruction of the superficial layer of the joint capsule is greatly facilitated and so is that of the tendon of the supraspinatus and the deeper, thicker portion of the capsule and of the capitular articular cartilage when it comes into contact with the acromion. This does not imply, however, that the articular cartilage on the head of the humerus may not be worn away elsewhere, for this frequently is the case.

After destruction of the upper portion of the articular capsule and its included tendons, the deeper surface of the deltoid muscle will be

Fig. 5.—Right shoulder with the deltoid reflected upward, revealing a large oval defect in the tendon of the supraspinatus bordered by smooth, sharp-edged margins of the latter (*2*) and (*4*), below which lie longitudinal ribbons of the deeper portion of the articular capsule (*3*). A frayed superficial part of the capsule is indicated by *1*, and the frayed deltoid portion of the subdeltoid bursa, by *5*. Not all longitudinal trabeculae of the capsule were numbered; a very narrow one lies between *1* and *2*.

Fig. 6.—A left shoulder joint with a similarly frayed and trabeculated capsule and tendon of the supraspinatus. It should be noted that all the trabeculae are longitudinal in direction with respect to the tendon of the supraspinatus.

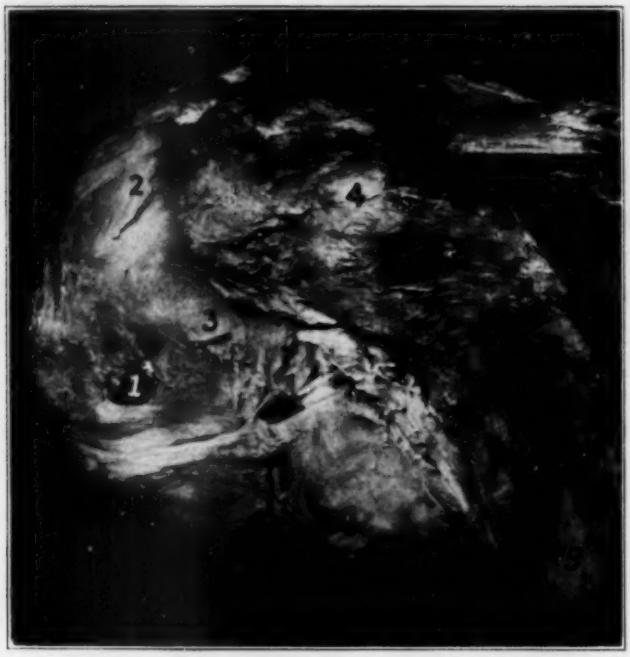

Fig. 7.—A photograph of the right shoulder from in front and above. Longitudinal slits are seen at *2* and *3* on either side of an unaffected tendon of the supraspinatus. A large, irregularly shaped hole is seen at *1*. The acromion is indicated by *4* and the clavicle by *5*.

Fig. 8.—Photograph of a right shoulder in a similar view showing a long longitudinal slit at the smooth anterior border of an unaffected supraspinatus tendon. Fine, narrow trabeculae, remnants of the superficial layer of the articular capsule, are seen bridging the slit.

exposed to similar wear, for under such conditions it must act vicariously for the destroyed portion of the capsule. A large part of the midsection of the proximal portion of the deltoid muscle may be destroyed completely. Whenever this happens, only the skin, the subcutaneous tissues, the superficial and deep fasciae and the fused sheaths of the deltoid muscle remain as a cover for the joint, and it would seem that it should be possible to diagnose such a condition in life. Such extensive destruction must be relatively rare, however, for I have seen but a single example in about a thousand bodies and have learned of no other instance, although fraying of a portion of the under surface of the deltoid muscle is not uncommon.

CHANGES IN THE LONG TENDON OF THE BICEPS

It is interesting that the intra-articular portion of the long tendon of the biceps muscle may be destroyed completely without destruction of the articular capsule and without the presence of pathologic changes anywhere in or about the joint. The tendon usually shows wear from beneath, but evidences of this not infrequently are present also on its upper surface. Erosion along the lateral margins may be due to a disproportion in width between the more proximal, intra-articular, portion of the tendon, which often is much wider, and the lateral walls of the bicipital sulcus, although it is more commonly due to contact with slight irregularities on the walls of the sulcus, apparently the result of periosteal injury from wear. Fraying of the under surface of the tendon usually results from contact with the cartilaginous margin of the head of the humerus, with the supratubercular ridge (see fig. 3, Meyer [2]), when present, or with slight irregularities on the floor of the sulcus, representing reactions to wear, or from contact with the lesser tuberosity in cases of partial or total dislocation. Whenever the relatively rough surface of the lesser tuberosity comes into contact with this tendon after partial or total forward dislocation of it and detachment of the capsular attachment between the lesser tuberosity and the sulcus, destruction of the tendon is greatly facilitated, but whenever it remains in a capsular sling it may become completely dislocated without grossly evident wear.

It is surprising and also very significant that the tendon of the supraspinatus and the long tendon may reach paper thinness without rupturing. Indeed, it is not at all uncommon to encounter long tendons of which so small a fasciculus remains that it cannot bear a weight of 10 pounds.[3] Yet I have never seen a case of acute rupture in the dis-

2. Meyer, A. W.: Spontaneous Dislocation and Destruction of Tendon of the Long Head of Biceps Brachii; Fifty-Nine Instances, Arch. Surg. **17**:493-506 (Sept.) 1928.

3. This no doubt is accounted for by the fact that secondary attachments are formed before the tendon is completely divided.

secting room, and I have never been able to find more than a proximal trace of the relatively long intra-articular portion of this tendon. This probably is due to division of it long before death and to destruction of the proximal portion by attrition and lysis.

An excellent specimen showing wear on both surfaces of the long tendon is represented in figure 9, which is a photograph of the intra-articular portion from the right shoulder of a man whose left tendon was wholly unaffected. The wear on the superficial or upper surface and on the anterior margin of this long tendon was due to contact with the overlying capsule—the so-called transverse humeral ligament.[4] Wear

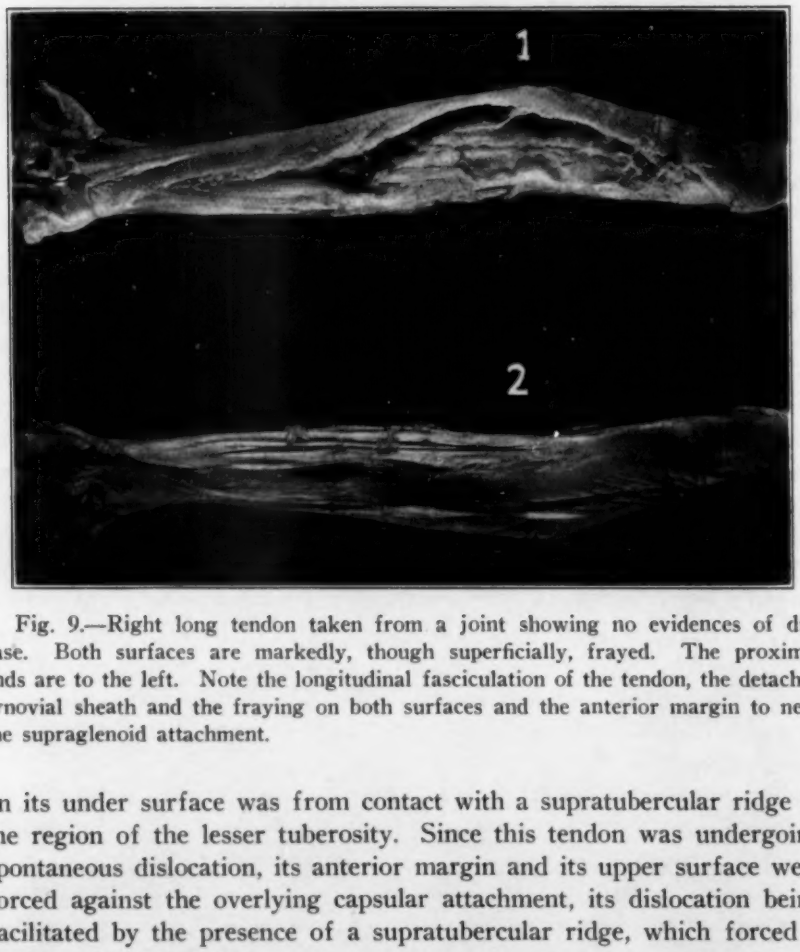

Fig. 9.—Right long tendon taken from a joint showing no evidences of disease. Both surfaces are markedly, though superficially, frayed. The proximal ends are to the left. Note the longitudinal fasciculation of the tendon, the detached synovial sheath and the fraying on both surfaces and the anterior margin to near the supraglenoid attachment.

on its under surface was from contact with a supratubercular ridge in the region of the lesser tuberosity. Since this tendon was undergoing spontaneous dislocation, its anterior margin and its upper surface were forced against the overlying capsular attachment, its dislocation being facilitated by the presence of a supratubercular ridge, which forced it

4. In cases of destruction of the overlying capsule, the superficial surface of the long tendon may also be worn by contact with the deep surface of the coraco-acromial ligament.

upward from the sulcus as well as forward. Under these circumstances, the tendon has to act as a wedge during its dislocation forward from the groove.

Whenever a portion of a tendon is deprived of the protective effect of the covering articular capsule and of its own sheath, its nutrition must of necessity be interfered with very seriously. At any rate, the portions of tendons so denuded become separated into longitudinal fasciculi of varying caliber. These readily become divided by contact with the cartilaginous margin of the head of the humerus or other surfaces of contact and may then easily be mistaken for portions of the tendon which were ruptured. This fasciculation of tendons is especially common on the under surface of the tendon of the subscapularis and of the tendons of the supraspinatus and the infraspinatus, but it occurs also on the upper or superficial portion of the tendon of the subscapularis and especially on the under surface of the long tendon of the biceps. I have encountered it in many places, even where the tendon of the peroneus longus glides on the facet of the cuboid bone, as shown in figure 10.

CHANGES IN THE TENDON OF THE SUPRASPINATUS

Fraying, thinning and final destruction of a part or the whole of the width and the thickness of the distal portion of the tendon of the supraspinatus as a rule are effected largely or wholly from without or above, while the tendons of the long head of the biceps and the tendons of the subscapularis and the infraspinatus usually are involved from within or beneath. However, the superficial portion of the tendon of the subscapularis may become fasciculated from movement against the under surface of a partially or wholly dislocated tendon of the biceps, after the protecting and overlying portion of the articular capsule has been detached or stretched to form a sling. Hence both surfaces of this tendon, like those of the biceps and of the supraspinatus, may suffer simultaneously, even if not equally.

Dislocation medially (forward) of the long tendon of the biceps may take place without detachment of the adjacent portion of the articular capsule, for the latter may merely stretch and form a sling which can glide on the long tendon in movements of the arm and protect it from contact with the underlying cartilage or bone. If the tendon of the subscapularis likewise retains its attachment, the long tendon may hence become dislocated completely without suffering much wear. But just as soon as the articular capsule becomes detached medially (anteriorly) and capitularly to the lesser tuberosity, dislocation of the intra-articular portion of the long tendon can begin; and it not infrequently becomes complete, although still in its normal position in the rest of the bicipital sulcus. Whenever the roof of the

intertubercular portion of the sulcus yields, the long tendon can gradually become dislocated forward over the lesser tuberosity and lie on the tendon of the subscapularis or on the lesser tuberosity itself, in case of detachment of the latter tendon from it. In the cases of the most maximal dislocation of the long tendon which I have seen, the more distal portion always lay in its normal position. This it does not leave either laterally or medially because of the firm attachment of the great tendons on either side and because of the directions of pull.

CHANGES IN CARTILAGES

The central area of the articular cartilage of the head of the humerus not infrequently also shows the effect of wear, but this does not imply that the central portion of the cartilage on the glenoid cavity must show the same effect, for a small portion of it is composed of fibroid instead of hyaline cartilage. The hyaline cartilage on the head of the humerus

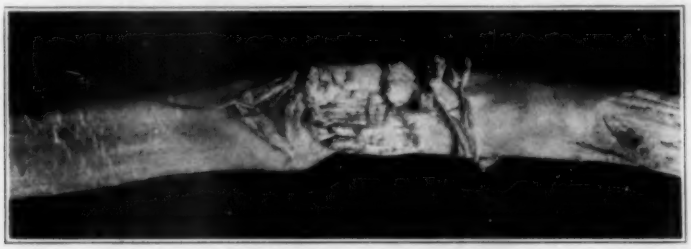

Fig. 10.—Right peroneus longus tendon seen from below with the distal extremity to the right. Note the detached synovial sheath near both extremities of the worn portion and the depth of the wear. The facet on the cuboid bone with which this worn area was in contact was normal in all respects and so were the rest of this tendon and its synovial sheath.

just proximal to the greater tuberosity may also be affected whenever it comes into direct contact with the under surface of the acromion, and it is in this region of the greater tuberosity and on the under surface of the acromion that polishing and eburnation are generally observed first. These polished areas seldom are congruent, however, and that is what one should expect. One may be several square centimeters in area, while the other coapted with it may be so small as to be scarcely noticeable, just as one can deface a large area with a fine diamond point.

The superficial membranoid layer of hyaline articular cartilage, which the older anatomists mistook for synovial membrane and which occasionally still is regarded so today, becomes frayed first. However, the thicker, uncalcified underlying layer not infrequently is chipped off from the underlying calcified layer in considerable areas over the head of the

humerus. Whenever the latter becomes detached, the underlying compacta is exposed, and polishing and eburnation can begin as soon as this area comes into contact with a similarly denuded area on the scapula or with an ossific plaque or nodule in the articular capsule. I have seen a number of such ossifications a centimeter or more in size which were exquisitely polished from contact with the head or the tuberosities of the humerus. Such nodules may also be polished on the external surface whenever they are in an area of the capsule which will permit them to come into contact with the acromion or the coracoid process.

Quite frequently the inferior acromioclavicular ligament and the respective portion of the capsule contain defects varying from small gaps to complete destruction of this portion of the capsule. Since shoulders with such defects which happened to come to my attention first also contained large defects in the upper portions of the humeroscapular articulations with complete division of the tendons of the supraspinatus muscle and partial destruction of the acromioclavicular ligaments, I had concluded that these defects in the acromioclavicular capsules and ligaments were due to contact with the heads of the humeri and the adjacent regions of the greater tuberosities. Of late I have observed a number of defects which cannot be accounted for in this way, however. In the latter instances the defects in the acromioclavicular capsule had been produced from above by contact with the inferior surface of the distal extremity of the clavicle and perhaps to some degree also by contact with the similar margin of the articular facet of the acromion. The lesion is surprisingly common and may be accompanied, preceded or followed, by the formation of a synovial bursa below this articulation in the connective tissue and fat overlying this portion of the supraspinatus muscle. Sometimes the musculotendinous junction is extensively fasciculated and frayed for an extent of several centimeters. Examples of these lesions with illustrations can be found in a forthcoming article on "Use Destruction in the Human Body," in *California and Western Medicine*.

Although in the long course of the gradual production of such lesions as these, slight hemorrhage may occur whenever a weakened structure suddenly yields, I have never encountered evidences of it. Thickened bursal walls and some coagulum were discovered only a few times in almost a thousand bodies, and effusions and adhesions were never seen. Gross and microscopic examination of a considerable series of specimens yielded no evidence of the presence of chronic inflammation.

Short distal stumps of the tendon of the supraspinatus muscle and of the adjacent capsule and transverse rents in the latter were seen only a few times. The defects and the fasciculations usually extended in the direction of pull, and the earliest capsular and tendinous defects

always were bounded by smooth, attenuated borders of capsule and tendon instead of by frazzled, thickened borders. Since, as shown in figures 7 and 8, longitudinal capsular defects also occur in the ventral (anterior) intertendinous portion of the capsule and broader defects also dorsally (posteriorly) beneath wholly uninjured tendons of infraspinatus muscles, these defects manifestly cannot be due to the rupture of associated tendons. The same thing is true of the fraying on the internal surface of the capsule opposite the cartilaginous margin of the head of the humerus and on the inner margin of the glenoid fibrocartilage, where fraying is practically universal. Rarely these cartilages may be nearly or wholly destroyed for a part of their extent.

The occasional presence of ossific nodules in different places in the upper half of the joint capsule, in the tendon of the infraspinatus muscle and even in the coraco-acromial ligament suggests that sudden trauma, greater in intensity than that which can be attributed to wear, may have been responsible for their production. That can readily be granted, for what laborer could be so fortunate as to escape sudden trauma to the shoulder during decades of an active life? The frank recognition of this fact surely cannot negate the overwhelming evidence against the assumption that all the lesions here considered result in this way or are the product of disease.

RUPTURE OF THE TENDON OF THE SUPRASPINATUS

Although Codman [5] (p. 120) thought weakness of the tendon of the supraspinatus muscle is due to its evolutionary history and spoke (p. 144) of "the strain on this little tendon," he nevertheless said (p. 150) that the insertion of this tendon is ¾ inch (1.9 cm.) long. On the basis of the best calculations available, the more prevalent form of tendon of the supraspinatus has a tensile strength many times that of its muscle belly. According to Triepel,[6] Fick,[7] Cronkite [8] and investigators mentioned by them, a supraspinatus tendon ¾ inch (1.9 cm.) wide and only 2 mm. thick could bear an estimated pull of over 700 pounds (318 Kg.), and one 3 mm. thick could bear a pull of over 1,000 pounds (454 Kg.). The muscle belly of the supraspinatus, on the other hand, even when it is large, has an estimated maximum

5. Codman, E. A.: The Shoulder: Rupture of the Supraspinatus Tendon and Other Lesions in or about the Subacromial Bursa, Boston, The Author, 1934.

6. Triepel, Hermann: Einführung in die physikalische Anatomie, Wiesbaden, J. F. Bergmann, 1902-1908.

7. Fick, Rudolf: Handbuch der Anatomie und Mechanik der Gelenke unter Berücksichtigung der bewegenden Muskeln, Jena, G. Fischer, 1910-1911, nos. 2 and 3; in von Bardeleben, K. H.: Die Anatomie des Menschen, Leipzig, B. G. Teubner, 1908, vol. 2, nos. 1 and 2.

8. Cronkite, Alfred Eugene: The Tensile Strength of Human Tendons, Anat. Rec. **64**:173-186, 1936.

strength of only 170.9 pounds (77 Kg.). Since the tendon of the supraspinatus muscle with the included capsule often is considerably thicker than 3 mm., the true disproportion between the strength of the unworn tendon and that of the muscle is greater than the foregoing estimates indicate. This alone makes rupture of a sound normal tendon by active muscle pull alone doubtful, and even if such a relationship did not exist and the tendon were relatively too weak and then were torn in part only, thus reducing its ability to withstand the next maximal muscular contraction, it then should yield suddenly. Moreover, since the worn supraspinatus tendon always becomes gradually thinner toward the point of its insertion, where it may be of membranoid thinness without perforation, it follows that all such tendons should have yielded earlier to sudden muscle pull and that they should contain transverse instead of longitudinal tears and trabeculae. And how could one entertain the belief that contraction of the subscapularis can avulse the large tubercular portion of the tendon, always leaving the relatively small diaphysial attachment intact, or that the greater upper part of the insertion of the infraspinatus may likewise be detached and the small, weaker, lower portion always remain unruptured? Yet this was always the case in my observations. Sudden rupture of the sound supraspinatus tendon should leave normally thick or thicker ragged, blunt ends, and sudden detachment of the tendon from its insertion should leave the fractured or intact border of the greater tuberosity protruding to impinge suddenly against the acromion in abduction of the arm. Since the cervical sulcus of the humerus sometimes is deep here and the tuberosity very prominent, such a sequence of events certainly would make itself felt severely during life and would not escape the attention of the clinician. Gradual erosion of the tendon, on the other hand, whether from within or from without, gives atrophy an opportunity to produce its leveling and reducing effects, lowering the height of the greater tuberosity and thus rounding the contour of this region of the humerus. Any one sufficiently familiar with the changes in it and in the region of the lesser tuberosity is aware of the great reduction sometimes suffered, especially by the latter, after detachment of the tendon. The entire place of the lesser tuberosity sometimes is occupied by a smooth, sunken field and at other times by a somewhat rougher field than normal. Examination of the spongiosa beneath may reveal a large absorption cavity akin toWard's triangle in the spongiosa of senile femurs (see fig. 1, Meyer[9]). I have never noticed equally marked atrophy of the spongiosa in the region of the greater tuberosity, but this may be due to the fact that since the latter continues to impinge

9. Meyer, A. W.: Unrecognized Occupational Destruction of the Tendon of the Long Head of the Biceps Brachii, Arch. Surg. **2**:130-144 (Jan.) 1921.

against the under surface of the acromion, the underlying spongiosa is continually subjected to strains sufficient to preserve it from the marked atrophy of disuse to which that underlying the lesser tuberosity is subjected after detachment of the tendon of the subscapularis.

THE QUESTION OF ATROPHY

I am aware of the fact that atrophy of the supraspinatus muscle has been emphasized as a diagnostic sign of rupture of its tendon, but I have been able to detect the presence of marked atrophy of it only a few times in cadavers. This applies likewise to the lateral belly of the biceps and to that of the subscapularis and of the infraspinatus. It appears to me that the absence of marked muscular atrophy, even in the presence of pronounced lesions in the tendons of these muscles, may be accounted for by the fact that the tendons are divided gradually, that the nerve supply of the muscles is left intact and that they hence can respond and continue to contract against some resistance. Moreover, it must not be overlooked that the long tendon of the biceps gradually obtains a secondary attachment in the region distal to the tuberosities, usually the lesser tuberosity, and that those of the subscapularis and infraspinatus never become wholly detached. The invariable presence of such a secondary attachment of the biceps, it seems to me, is adequate evidence that sudden rupture probably did not play a part in these cases, for were this so, considerable elongation of the brachial portion of the tendon should be common instead of very rare. Moreover, the invariable reattachment of this tendon at the same level on the humerus could not be accounted for. Reports of cases of acute rupture in which operative treatment was employed (Gilcreest [10]) confirm these conclusions. On the other hand, since the tendon must yield gradually when weakened by wear, it obtains a secondary attachment before it is completely divided.

I have never seen any evidences of the existence of osteitis in the region of the lesser tuberosity, as described by Béra,[11] and do not believe that it could be a factor in reducing the size of the lesser tuberosity and thus facilitate dislocation of the long tendon of the biceps. Chronic osteitis, unless suppurative, should not cause destruction of the

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10. Gilcreest, Edgar Lorrington: (*a*) Rupture of Muscles and Tendons, Particularly Subcutaneous Rupture of the Biceps Flexor Cubiti, J. A. M. A. **84**:1819-1822 (June 13) 1925; (*b*) Two Cases of Spontaneous Rupture of the Long Head of the Biceps Flexor Cubiti, S. Clin. North America **6**:547-554, 1926; (*c*) Dislocation and Elongation of the Long Head of the Biceps Brachii: An Analysis of Six Cases, Tr. West. S. A. **44**:260-296, 1934.

11. Béra, Albert: Du syndrome commun à la rupture, à la luxation et à l'élongation du tendon du long biceps, Thèse de Paris, no. 304, Paris, Jouve & Cie, 1911.

bone, and as long as the ligamentous, capsular and tendinous attachments remain intact, dislocation of the long head is impossible. The following words from Béra's splendid thesis deserve repetition, however:

Les anomalies congénitales.—Il est intéressant de faire remarquer que les anatomistes ont fréquemment cité des cas où la longue portion du biceps se fixait anormalement dans la coulisse bicipitale, sur les tubérosités humérales, sur la capsule de l'articulation de l'épaule (Dr. Le Double, *Variations du système musculaire de l'homme,* t. II). Il est permis de se demander si certains de ces faits ne rentrent pas dans le cadre de l'affection qui nous occupe; et si cette anomalie d'insertion n'est pas souvent l'effet secondaire d'un traumatisme plutôt que le résultat d'une malformation congénitale.—Ici encore, la perceptibilité du tendon bicipital au-dessous du bord deltoïdien, constatée sur le vivant ou sur le cadavre, permettrait de trancher la question [p. 48].

Cette luxation primitive et isolée existe-t-elle en tant qu'affection franche et persistante, la chose est peu probable. Elle entraine non seulement l'ouverture de la coulisse bicipitale, mais encore la destruction de la capsule articulaire et du tendon du sus-épineux [p. 38].

Congenital Anomalies.—It is interesting to note that anatomists have frequently cited cases in which the long head of the biceps muscle was abnormally fixed in the bicipital groove, on the humeral tuberosities or on the capsule of the joint of the shoulder (Dr. Le Double: Variations du système musculaire de l'homme, vol. 2). It is allowable to ask whether some of these facts do not enter into the picture of the disturbance of which we treat and whether this anomaly of insertion is not often the secondary effect of a traumatism rather than the result of a congenital malformation. Perception of the bicipital tendon below the deltoid border in the living subject or in the cadaver would allow one to decide the question [p. 48].

That this primary and isolated luxation exists as a frank and persistent disturbance is improbable. It leads to not only the opening of the bicipital groove but also the destruction of the joint capsule and of the tendon of the supraspinatus muscle [p. 38].—Publisher's translation.

I have never seen a case of spontaneous dislocation of the long tendon dorsally over the greater tuberosity, and it seems to me that this is wholly precluded by normal anatomic conditions. All dislocations of the biceps tendon seen by me were forward dislocations, and I never saw any evidences that fractures or previous dislocations had been contributory factors in producing the lesions I am considering. However, that there may be such cases I gladly recognize.

Whenever the tendon of the long head is retracted into the brachium somewhat from yielding of the secondary attachments before, or from retraction of the tendon after, rupture of the weakened intra-articular portion, the brachial part of the tendon is longer, and marked asymmetry between the position of the two muscle bellies of the biceps necessarily results and is intensified whenever the lateral belly atrophies because of elongation of the tendon.

No one sufficiently familiar with the evidence present in cadavers with respect to these lesions of the shoulder and others present through-

out the entire human body can have any doubt as to their significance. Indeed, the only phenomenon which still perplexes me is the exact manner of detachment of the subscapularis tendon from its insertion to the lesser tuberosity, for the middle portion of this tendon may be detached when the rest of it still remains attached by a peripheral ring of fine cylindric tendinous fasciculi, and complete detachment of the tendon from the lesser tuberosity may be present in cases of the developmental absence of the lateral belly of the biceps brachii. In some instances, too, only a few thin ribbon-like bands of tendon remain attached at intervals from above downward on this tuberosity. Such cases as these are rather puzzling, but they are that no matter what one may regard as causative factors in detachment of this tendon. Since atrophy of the tuberosity occurs in the areas of detachment of the tendon, it is possible that this absorption plays a rôle in the production of these anomalous detachments. Moreover, since the blood supply of tendons comes almost wholly from without, it follows that the destruction of the enveloping sheath must affect the firmness of attachment of the tendon in the course of time. Sharp or irregular portions of the cartilaginous margin of the head of the humerus, the osteogenetic reactions evoked in the traumatized periosteum and the absorption of cartilage no longer subjected to pressure from the tendon of the long head, all may be factors in the production of these detachments of the tendon of the subscapularis. Although the lesser tuberosity sometimes is normal in size and smoothness and covered with a thickened periosteum after the detachment of this portion of the insertion of the tendon of the subscapularis, it may be decidedly pitted and covered by an exceedingly thin periosteum or show little evidence of atrophy and be roughened by numerous low exostoses.

I have often wondered whether dislocations of the shoulder are not commoner in cases of maximal destruction from attrition and whether in some cases habitual dislocation may not be due to it, but I never have encountered such a condition in the dissecting room. However, since the stability of the shoulder joint depends so largely on muscles and tendons, it would seem that partial or complete destruction of the tendon of the supraspinatus and partial destruction of that of the infraspinatus accompanied by loss of the tubercular attachment of the subscapularis tendon must seriously reduce the stability of the joint even if not markedly affect its functional activity.

It seems that it should be possible to notice the presence of bony contacts in all cases in which this has become possible in consequence of capsular and tendinous or cartilaginous attrition. From my experience in auscultation of various supposedly normal articulations during movements which required no special strain, I am led to believe that

more can be made of this method of examination. It would also seem that the gap between the acromion and the head of the humerus indicated in roentgenograms must be reduced whenever the capsule or the articular cartilage on the head of the humerus or both are destroyed. Under the relaxation of deep general anesthesia, it should also be possible to produce a tap by forcing the head of the humerus against the lower surface of the acromion in all cases of large capsular defects. Although functional disabilities may be very misleading, some of them nevertheless must be suggestive with regard to both the extent and the character of the lesions present.

It is true that the literature on conditions of the shoulder plainly suggests that some of the symptoms, signs and disabilities accompanying the trauma of attrition have been attributed to dislocation, rheumatic arthritis, arthritis deformans, rupture, neuritis, periarthritis, bursitis, tendinitis, senile necrosis, wear following disease and the effect of sudden motion, but that is an inference not based on personal knowledge of the cases concerned. The implication is not, to be sure, that none of the conditions mentioned can cause changes in and about the shoulder joint but that there may be profound changes without the presence of any of the foregoing conditions except that of wear.

REVIEW OF LITERATURE

Any one who has dissected a series of human bodies or has carefully observed their dissection must have encountered some, even if not all, of the foregoing lesions. Indeed, there is ample evidence in the medical literature to show that occasional observers have tried to explain very similar even if not identical phenomena, although Smith [12] thought that they have been "entirely overlooked . . . from inadvertency or other causes . . . in the ordinary dissection of bodies." During the dissection of forty bodies, Smith observed seven instances of destruction of the upper part of the capsule of the shoulder joint, destruction of part of the long tendon of the biceps and detachment of the tendon of the subscapularis and concluded that they were due to external factors such as a blow or fall, but he thoughtfully added:

> It is a curious fact, that in two instances both joints of the same individual should have presented nearly similar appearances, and that the same cause should probably have produced exactly similar results [p. 222].

According to Smith:

> The separation of the tendon of the subscapularis muscle from the lesser tubercle, (excepting in one case), where it was only partially torn away, may

12. Smith, John G.: Pathological Appearances of Seven Cases of Injury of the Shoulder Joints, with Remarks, Quart. Periscope, Am. J. M. Sc. **16**:219-224 (May) 1835; London M. Gaz. **14**:280-285, 1834.

be looked upon as the common result of the accident which produced these appearances; not so, however, with the tendons of the supra and infra-spinatus, and the teres minor muscles, from the greater tubercle, which appear to be more uncertain. In two instances all three tendons were torn away; in three cases only the tendon of the supra-spinatus; and in the remaining two cases, the whole of the tendons preserved their natural attachments [p. 222].

Callaway,[13] like Smith, stated explicitly that what he observed were cases of traumatic rupture and dislocation of the tendon of the long head of the biceps muscle, and Rapp,[14] who used the significant and appropriate word *Usur* in connection with lesions of the tendon of the long head of the biceps and who also represented the proximal portion of the torn tendon with a brushlike, free extremity as shown in figure 11, regarded this as due to growth after injury and concluded that luxations, disturbance of the circulation in the tendon sheath and inflammation may cause changes in the normal tendon of the biceps such as he encountered and the significance of which he was considering.

One of the historically most interesting and significant discussions of conditions in the shoulder is presented in an article by Robert Adams,[15] which appeared in Todd's "Cyclopaedia of Anatomy and Physiology." Under the subhead of "chronic rheumatic arthritis of the shoulder joint," a "slow chronic disease" which "itself never causes death," Adams described many lesions such as those considered here, including destruction of the upper portion of the articular capsule; division, dislocation and reattachment of the long head of the biceps muscle; local destruction of the articular cartilages; destruction of the deeper portion of the capsule of the acromioclavicular articulation, and polishing of the head and tuberosities of the humerus and of the under surfaces of the acromial and coracoid processes. However, Adams also reported and included enlargement of the bones of the shoulder; thickening and enlargement of the articular capsule with adhesions to the acromion, coracoid process and the head of the humerus; frequent complete transverse division of the acromion within an inch of its extremity; effusions into the articular capsule with the presence of osseous fragments, and dislocation of the head of the humerus upward beyond the level of the clavicle and acromion and "completely backward on the dorsum of the scapula." The illustrations accompanying Adams'

13. Callaway, Thomas: A Dissertation upon Dislocations and Fractures of the Clavicle and Shoulder-Joint, Jacksonian Prize Essay for 1846, London, S. Highley, 1849, p. 178; reviewed, Lancet **2**:400-401, 1849.

14. Rapp, Hermann: Ueber die Usur der Sehne des langen Kopfes vom M. Biceps brachii, Dissert., Tübingen, 1866.

15. Adams, Robert: Shoulder Joint, in Todd, R. B.: Cyclopaedia of Anatomy and Physiology, London, Longman [and others], 1852, vol. 4, pp. 571-621. Although this volume appeared in 1852, the text plainly implies that the article was written in 1848.

article show the presence of extensive articular exostoses and pronounced osseous extensions, such as are seen in so-called arthritis or arthrosis deformans. He emphasized the presence of "rheumatic arthritis" in practically all joints in his cases and believed that the case of Cruveilhier, in which ". . . the external extremity of the clavicle and the neighboring part of the acromion were in a great part destroyed," likewise belonged in the same category. Adams thought that "absorption" of the articular cartilages, both hyaline and fibroid, was effected by contact with capsular fimbria or so-called synovial villi, but recognized the presence of friction under such pathologic conditions.

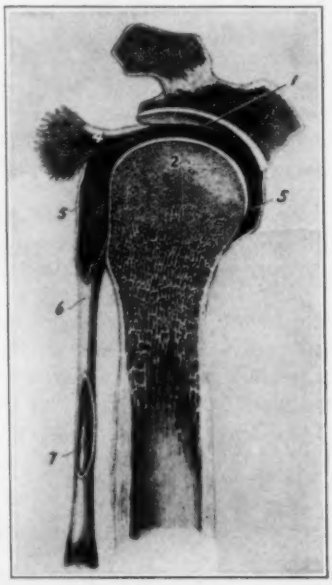

Fig. 11.—Sagittal section of a right shoulder. The glenoid cavity of the scapula is indicated by *1*, the head of the humerus by *2*, the tear and partly an outgrowth of the upper end of the tendon of the long head of the biceps by *3*, the long head of the biceps by *4*, the synovial capsule of the shoulder by *5* and the tendon sheath of the long tendon of the biceps by *6*. The last-mentioned structure contains a sequestrum of the original tendon, indicated by *7*. Those familiar with attrition effects will recognize that *3* is due neither to tear nor to growth. (After Rapp.[14])

He thought, however, that "no doubt some few cases of chronic rheumatic arthritis of the shoulder joint in the living and in the dead have been mistaken for *partial dislocation of the head of the humerus, the results of accident* . . . " He was ". . . convinced that all these phenomena combined should by no means be considered as proof of any accident having occurred to produce them; but, on the contrary, should be looked upon as the usual result of chronic rheumatic arthritis

of the shoulder." He emphasized that in 1836 he had called attention to the fact that appearances delineated by Sir Astley Cooper in his treatise on "Fractures and Dislocations" corresponded exactly to the appearances found in the cases of "chronic rheumatic arthritis" and that all his subsequent experience "amply confirmed" this opinion, adding that he ". . . thought it necessary to enter into this subject thus minutely, because we are convinced that, up to the present hour, these remarkable appearances, when met with, have been misunderstood even by some of the most intelligent anatomists and physicians."

In the well known "Treatise on the Human Skeleton Including the Joints" by Humphry,[16] some similar lesions were likewise mentioned, for when considering defects in the long tendon of the biceps, Humphry wrote:

> The rent most often happens about half an inch from its glenoid attachment; and is most frequent in elderly persons, in whom the tendon becomes thin, in whom the synovial fluid is rather scanty, and in whom other changes, attendants on chronic rheumatism, are likely to be in progress.

I do not know of any evidence in support of the assumption that tendons become thin and synovia scanty, under the conditions concerned here, but Humphry very significantly said:

> Under these circumstances there can be no doubt that the tendon sometimes becomes *worn through* by the friction of the head of the humerus upon it; and I quite agree with the writer in the Cyclopedia of Anatomy [Adams], that many of the specimens, in which the biceps tendon stops short in the bicipital groove, or becomes lost in the capsule of the joint, are to be attributed, not to rupture of the tendon, but to the continued effects of chronic rheumatism, originating in an accident, or commencing spontaneously [p. 415].

It is noteworthy that Humphry added the following in a footnote:

> It would appear, from some dissections of the shoulder-joint described by Mr. J. G. Smith, *Medical Gazette,* XIV. p. 280, that, not only may the tendon of the biceps be worn through, but that the capsular ligament and the tendons of the supra- and infra-spinatus muscles may undergo the same change, and the sub-deltoid bursa thus become opened into the cavity of the joint. In several of the cases mentioned the sub-scapularis tendon was more or less detached from the lesser tubercle. Mr. Smith regards these changes as the result of accident; but several other points in the state of the joints, and the existence of similar conditions in both shoulders of the same person, render it more probable that they were the gradual result of friction and chronic rheumatism.

It is of interest in this connection to recall that Sir W. Arbuthnot Lane[17] thought ". . . that the pressure which in elderly subjects

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15. Humphry, George Murray: A Treatise on the Human Skeleton Including the Joints, Cambridge, Macmillan & Co., 1858.

17. Lane, W. Arbuthnot: A Remarkable Example of the Manner in Which Pressure-Changes in the Skeleton May Reveal the Labour-History of the Individual, J. Anat. & Physiol. **21**:385-406, 1887.

or in those of low vitality destroys the articular cartilage and the subjacent bone, rendering the latter eburnated, and determining the formation of additamentary bones, produces in a young subject a similar alteration in the form of the articular surface, without affecting the vitality or continuity of its cartilaginous covering" (p. 386). He thought that ". . . pressure-changes in the skeleton may enable us to detect the habits and labour-history of the individual" (p. 386) and concluded regarding one case that "during the whole of his active life this man had been continually occupied in shovelling some material which was very coarse . . ." (p. 398). Sir Arbuthnot also spoke of a case in which ". . . the glenoid ligament was separated completely from the upper third of the margin of the glenoid cavity, being connected to it indirectly by a thin membrane. The upper part of the glenoid cavity was slightly increased in area. The cartilage covering the lower part had been converted into synovial membrane and areolar tissue . . " (p. 388). He attributed these things to an "upward displacement of the head of the humerus which ensues in old age. . . ." (p. 388), but he recognized that bone may be removed as a result of "friction and pressure" and thought that pressure from the radial head of the humerus on the head of the radius may cause polishing and eburnation (p. 389). He offered this explanation also for perforated or wholly destroyed articular cartilage at the distal extremity of the ulna (p. 390) and believed that he had "shown that constant pressure exerted by the inferior articular processes of the 4th lumbar vertebra, and by the superior articular processes of the sacrum upon that portion of the arch of the 5th lumbar vertebra which lies between their extremities, may cause its partial or complete section" (p. 398). After considerable discussion of this matter, Lane referred to two cases of this anomaly reported by Professor Neugebauer, which the latter said were very puzzling and which he ascribed to congenital causes (p. 400). Although Lane also referred to an article by Sir W. Turner in which the latter described "several cases of want of union of the neural arch with the body of the 5th lumbar vertebra, which he ascribes to imperfect development" (p. 401), Lane nevertheless held to his opinion as to the origin of these anomalies and said ". . . *that the causation of the condition is pressure and not fracture, or a non-union of bony centres*" (p. 401).

These quotations pointedly reveal the significance of the older literature, in which a fall, blow, fracture, dislocation or disease, especially arthritis, is usually assigned as cause for the lesions encountered. It is largely for this reason that I have not discussed that literature fully, for it ill becomes one who did not even see the cases described by others

to question the correctness of their interpretations. A fine summary of the extensive older literature is given by Gilcreest.[10e]

Codman, who has given so much attention to painful shoulders, thought that a hole in the humeroscapular capsule might close completely and stated [18] that he put a good many silk "sutures à-distance" between the separated ruptured ends of a supraspinatus tendon, because ". . . it seemed possible for repair to take place along the silk sutures." No one will deny that a rupture in a normal capsule or tendon may heal provided the torn ends are in direct or indirect apposition and are put at rest immediately, but Dr. Codman stated that he never operated in any case until three weeks after rupture, and since the period given apparently was inferred there is no assurance that it may not have been three months after. Moreover, I infer that he believes that defects which are the result of wear, such as I am considering here, also may heal, for he said [5] (p. 86) that the triangular defects in the supraspinatus tendon always become rounded, as does a defect in the epithelium of the skin, "before it heals" and that incomplete ruptures (those in which the rent in the tendon is from ¼ to ½ inch [0.6 to 1.3 cm.] in length) will heal of themselves. Codman had also suggested earlier that clipping away the "inflamed" bursa over the ruptured tendon would cause satisfactory healing, but he [5] later doubted this (p. 131).

Wilson [19] considered only cases of complete rupture of the supraspinatus tendon with gaps up to "2½ inches in diameter." [!] He spoke of the presence of "fibrillation of the tendon and shredding with the formation of irregular tabs of tissue, sometimes calcified," and "occasionally fibrillation" of the articular cartilage on the head of the humerus as being most evident in patients in whom the longest time had elapsed since injury. Wilson found no pathologic changes present at the time of operation but said that the greater tuberosity is usually "bare" of tissue. If so, it follows that the distal stub of the ruptured tendon must have been completely resorbed in the interval between the accident and the operation, and if the greater tuberosity really was completely bare, it also is necessary to account for the absence of periosteum, which cannot be attributed to atrophy. On referring to some lesions of the shoulder reported by me and the suggested explanation for them, Wilson correctly said that "Most of his investigations have been made on dissecting room subjects," but he nevertheless added that these ". . . must have been cases of rupture of the supraspinatus tendon." [!]

18. Codman E. A.: Complete Rupture of the Supraspinatus Tendon: Operative Treatment with Report of Two Successful Cases, Boston M. & S. J. **164**:708-710, 1911.

19. Wilson, Philip D.: Complete Rupture of the Supraspinatus Tendon, J. A. M. A. **96**:433-439 (Feb. 7) 1931.

Although Keyes [20] found almost complete obliteration of the subdeltoid bursa by fibrous adhesions which "resembled" those "in the peritoneal cavity, particularly in the pelvis of the female," and mentioned the presence of hypertrophic bony changes on the greater tuberosity and the adjacent regions, of deposits of calcium in the supraspinatus tendon, of increased fragility of the bone of the greater tubercle and of erosion of the articular surface of the humerus, he nevertheless emphasized that fraying of the tendon of the long head was not observed and that the tendon was intact in every instance, but in 1933 he [21] said that all his cases "were associated with a certain amount of tendon fraying and bony irregularity" and assured readers that "this description of one lesion covers the main features of all the other . . ." of a series of fourteen of seventy-two cadavers, or nineteen of one hundred and forty-two shoulders. A larger series of observations would undoubtedly have revealed greater variations among them.

Keyes also said, in 1933, that the tendon of the supraspinatus muscle "during the terminal half of its course [6 cm. according to Keyes] . . . is inseparable from the joint capsule," and that "its two borders may be distinguished from the adjoining borders of the tendon of the infraspinatus and subscapularis only with some difficulty. . . ." He added that rupture of the supraspinatus tendon "is related to subdeltoid or subacromial bursitis;" that the lesions considered by him were due to "some process . . . which progressed with years, gradually causing degeneration first of the floor of the subacromial bursa, then wearing through the tendon at its insertion into the greater tubercle, and finally causing a perforation or rupture of the tendon." However, in 1935 Keyes thought that the lesions were "traumatic and degenerative in nature" and that "age, long use, and trauma seemed the chief causative factors," although "alterations in tissue growth and metabolism were believed to act locally . . . ." He added that infection caused the changes seen in few of these shoulders. Keyes also reported the absence of atrophy of the supraspinatus muscle "even when a large rupture [up to 2.2 cm. wide by 3.5 cm. long] was present in its tendon . . . ."

Any one who has microscopically or even macroscopically examined the borders of the capsular defects I am considering must realize that spontaneous healing is absolutely out of the question, no matter what the size of the defect or the degree of use of the arm, for in order that healing may occur it is necessary not only that the edges of the defect

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20. Keyes, E. Lawrence: Anatomical Observations on Senile Changes in the Shoulder, J. Bone & Joint Surg. **17**:953-960, 1935.

21. Keyes, E. Lawrence: Observations on Rupture of the Supraspinatus Tendon, Based upon a Study of Seventy-Three Cadavers, Ann. Surg. **97**:849-856, 1933.

be freshened and brought together but that the structures be put at rest. The mere presence of fluid or even of a coagulum between the widely separated ends of tendons divided by attrition weeks and months previously would not evoke or facilitate the least reparation and the startling suggestion that such reparation might occur along silk sutures between the widely separated ends of a tendon or capsule does not seem to rest on a sound basis.

I am reminded that Codman and Akerson,[22] writing on "The Pathology Associated with Rupture of the Supraspinatus Tendon," said that it is the usual cause of traumatic subacromial (subdeltoid) bursitis. I am at present writing a book to support this contention [p. 353]. . . . The large number of bilateral cases (fourteen) found at autopsy speaks against traumatism and in favor of some form of slow necrotic destruction" [p. 356]. It is important to note that Codman and Akerson apparently found no pathologic condition responsible for the alleged rupture of the tendon of the supraspinatus, and since bursitis is assumed to follow its rupture it manifestly cannot be a factor in its production. The only other pathologic process postulated is a "slow necrotic destruction" for the occurrence of which Codman and Akerson seem to have found no postmortem evidence except the presence of alleged rupture which the necrotic changes were supposed to cause.

It is important to note that Codman and Akerson gained their information on cadavers through "a cut . . . made on the anterior aspect of the shoulder-joint from the acromioclavicular articulation downward for about two inches" (p. 352). Any one who has followed careful dissections of the shoulder will recognize how inadequate such a method of postmortem examination is for revealing the true conditions in the joint. I admit that some one may examine a single shoulder thoroughly and come to a correct conclusion regarding the condition but scarcely on evidence obtained from inspection through a 2 inch (5 cm.) incision at autopsy.

I am wholly at a loss to understand how these authors could hold that "a purely anatomic argument against Doctor Meyer's theory is that the impingement of the head of the humerus on the lower surface of the acromion does not occur in the normal individual" (p. 357), for I certainly implied nothing of the sort. It does not require a lifetime in the dissecting room to apprise one of the fact that the head of the humerus is not in direct contact with the acromion when the joint capsule is intact! Moreover, the two sentences which follow the one just quoted are equally perplexing and unintelligible, for Codman and Akerson wrote: "The friction at this point is taken up by the outer

22. Codman, Ernest A., and Akerson, Irving B.: The Pathology Associated with Rupture of the Supraspinatus Tendon, Ann. Surg. **93**:348-359, 1931.

side of the tuberosity which is covered with the lower surface of the bursa. Furthermore, the coraco-acromial ligament partially intervenes" —evidently between the head of the humerus and the acromion! It is difficult to see why the shoulder joint ". . . is anatomically not likely to show the usual changes associated with arthritis" (p. 356) and that "from our understanding of accepted principles of pathology, such actual destruction by friction is well nigh impossible" (p. 357).

These authors further stated that "a feeble effort at repair" may result in ". . . the formation of a falciform edge (in the vertical plane) of fibrosynovial tissue which smooths the edge of the rupture around the whole circumference [pp. 354-355] . . . The rents in the other tendons extend laterally from that in the supraspinatus, even to the extent of evulsion of all of the tendons of the short rotators" [p. 356]. Certainly, no one will deny that all the tendons of the short rotator muscles may be evulsed, but surely not without a history of serious trauma and recourse on the part of the patient to immediate treatment, thus throwing the condition wholly outside of the category of those that I considered and attributed to attrition.

It is noteworthy that Codman, who has given so much and such long attention to these matters in living subjects, said in 1934 [5] (p. 84) that although he enjoyed the cooperation of friends he never saw a fresh rupture of the supraspinatus tendon on the operating table or at autopsy and never operated on a *young* man and "demonstrated" the presence of a ruptured tendon. The average age in his series of patients with assumed complete rupture of the supraspinatus tendon was 55, and of partial rupture, 49 years! The fact that none of the cases of so-called rupture mentioned by Codman occurred before the age of 50 and that none came to his attention for weeks or months after the presumed rupture surely is of great significance and cannot be explained by Codman's belief that partial acute ruptures are painless.

Codman regarded all communications between the subdeltoid bursa and the joint cavity as "pathologic" and suggested arthritis, periarthritis, bursitis, chronic inflammation and possibly overuse as causes for rupture of the tendon of the supraspinatus muscle. Yet he expressed the belief (p. 97) that rupture of this tendon is usually caused by sudden elevation of the arm, not under a load but merely in order to maintain body balance when in a drunken state! This idea was emphasized further on page 134, where it is said that "adequate injury—usually a fall" is responsible for the rupture.

Although bony excrescences were present in the case shown in his figure 3, plate 4, Codman stated that he found no evidences of pathologic changes, but on page 108 it is stated that he now believes that trauma and arthritis play alternating parts and (p. 109) that "some toxic

disturbance" may render the "joint tissues and the adjacent tendons sub-normal, and even partially necrotic, so that slight traumata which would not affect a normal tendon, easily tore their fibers." On page 112 of this same work it is said that:

> . . . it is fair to postulate that most of the operative cases had some degree of "arthritic" degeneration in their musculo-tendinous cuffs before their accidents (p. 112). . . . When I first began my work on "Stiff and Painful Shoulders," the usual diagnosis assigned to such cases was "periarthritis." Following my articles drawing attention to the anatomic characteristics of the bursa and the importance of its recognition, the term "subdeltoid bursitis" replaced that of "periarthritis," and since my second paper, the adjective "subacromial" has largely replaced that of "subdeltoid" (p. 119); . . . we might say with much truth that there is no such condition as a chronic arthritis *per se* of the shoulder, for, owing to the structure of the joint, it would always be a periarthritis; . . . I contend that the defects in this cuff so frequently found at autopsy were originally largely traumatic, although unhealthy tendon may have suffered the trauma (pp. 117-118), . . . tendinitis both of the calcified and uncalcified forms precedes the peaks of incidence of the ruptures, partial and complete, by about five to ten years (p. 138).

According to Codman (p. 122), complete rupture of the supraspinatus tendon causes a chronic bursitis, and inflammation of the tendon probably remains painless until a subacromial bursa becomes involved. He further expressed the belief (p. 121) that hyperabduction of an arm for a period of one to two hours may cause a bursitis which may last for half a year; that [23] the strain on the subdeltoid bursa in operations on the breast may cause bursitis; that this is the rule rather than the exception, and that "brachial neuritis" usually is a bursitis.

Excellent evidence against the assumption that the lesions I have considered are pathologic in origin is afforded by the cartilaginous changes observed by Glauning [24] in the knee joints of horses. Except in two respects, these were wholly comparable to those found in the human being, and Glauning stated that none of the animals had been lame and that the changes observed were not due to inflammatory conditions. This is in accord with the findings in worn human tissues from the dissecting room which likewise revealed no evidences of disease.

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23. Codman, Ernest A.: Rupture of the Supraspinatus Tendon, Surg., Gynec. & Obst. **52**:579-586, 1931.

24. Glauning, Werner: Altersveränderungen im Kniegelenk des Pferdes, Inaug. Dissert., Habbel, Regensburg, 1936.

INFLUENCE OF LAPAROTOMY ON THE GASTRIC MOTOR MECHANISM OF MAN

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Disturbances in the motility of the stomach are frequently encountered after laparotomy. These may become of serious import if the underlying morbid process is not corrected. In this respect an understanding of the perverted physiologic activities of the human stomach is of peculiar importance, for without it therapy is uncertain and may prove to be unsuccessful.

To investigate certain of the problems which arise concerning pathologic gastric physiologic activity, preliminary observations were made on the influence of the extrinsic innervation on the motility of the human stomach. Unusual opportunities were available for studying the effect of resection of the splanchnic and of the vagus nerves on the gastric motor function. The surgical indications, the apparatus used and an analysis of the results obtained have already been reported.[1] The results of these fundamental observations led us to make an analysis of certain abnormal gastric manifestations so frequently encountered after laparotomy. The purpose of this paper is to report the results of this investigation.

METHOD OF INVESTIGATION

Gastric motility was studied in seven patients. Two presented cholecystitis associated with cholelithiasis for which cholecystectomy was eventually performed with the patients under nitrous oxide-ether anesthesia. Five were operated on for inguinal hernia. In this group spinal anesthesia was used for four patients and nitrous oxide-ether anesthesia for the fifth.

In the first group both patients presented the findings characteristic of disease of the biliary tract. Roentgenographic studies revealed in one patient numerous

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1. Barron, Louis E.; Curtis, George M., and Haverfield, William T.: The Effect of Bilateral Resection of the Splanchnic Nerves on Gastric Motility in Man, Arch. Surg. **32**:577 (April) 1936. Barron, Louis E.; Curtis, George M.; Haverfield, W. T., and Lauer, B.: Effect of Vagotomy on the Gastric Motor Mechanism of Man, ibid. **34**:1132 (June) 1937.

opaque shadows suggestive of facetted gallstones. In the other there was nonvisualization of the gallbladder after the intravenous administration of the dye. The diagnosis of cholecystitis associated with cholelithiasis was confirmed for both patients at operation.

Tracings of the gastric motility were made during prolonged preoperative control periods. Observations were resumed as soon as postoperative nausea and vomiting subsided. This was approximately four days after cholecystectomy.

In the second group one patient presented bilateral indirect inguinal hernia. Herniorrhaphy was performed in two stages. Tracings of the gastric motility were made during the preoperative control period, during the first operation and throughout an intervening ten day period and were resumed subsequent to the second operation. It thus became possible to compare the results of two separate but similar operations on the same patient. Since spinal anesthesia was employed for four patients, tracings of the gastric motility were possible during the actual hernial repair as well as immediately after the surgical procedure. Daily observations were continued on all patients until they were discharged from the hospital.

Gastric motility was investigated by the balloon and kymograph method. The specific details of this procedure and the manner of ascertaining the position of the balloon in the stomach, as well as the method of analyzing the results, have been described.[1] Observations were made in the morning about fourteen hours after the evening meal. The usual duration of each continuous observation was about five hours. Tracings of the gastric motility were also made on the morning of operation to note any disturbances in motility possibly associated with emotional changes resulting from anticipation of the surgical procedure.

The specific criteria for the evaluation of gastric motility were essentially the same as those described in the earlier reports.[1] The data obtained from each patient were similarly arranged in tabulated form to facilitate accurate comparative study. Those appearing in this paper are representative. In a few instances a signal magnet and electric key were incorporated into the system. The patients were instructed to indicate any abdominal discomfort by pressing the key, which in turn produced an informative mark on the tracing, thereby making it possible to note any correlation between the abdominal symptoms and the results appearing on the kymographic record.

RESULTS

When the results of this investigation were surveyed in their entirety, it was evident that variation was present. It would seem that the specific disease, its duration and the eventual surgical management were responsible, at least in part, for this. As a consequence, the results are presented with reference to the primary disease and its subsequent therapy.

Cholecystitis with Cholelithiasis.—During the preoperative control period the duration of periods of motility was somewhat increased (table 1). In many instances they persisted for more than one hundred minutes (fig. 1). Frequently mild epigastric pain was experienced during this prolonged period of gastric activity. By means of the electric key and signal magnet, it appeared that this abdominal discomfort accompanied intense gastric contractions.

TABLE 1.—*Analysis of Experiments on Gastric Motility During the Control Period in a Case of Cholecystitis with Cholelithiasis*

Date of Experiment	Duration of Experiment	Periods of Quiescence		Periods of Activity		Contractions					Comment
		No.	Duration	No.	Duration	Type	No.	Duration	Interval Between	Amplitude	
5/25/35	4 hr.	..	.....	1	29 m.	II	23	Max. 55 s.* Min. 20 s.	Max. 3 m. Min. 1 m.	Max. 5 cm. Min. 2.5 cm.	
		1	25 m.	1	77 m.	II	46	Max. 50 s. Min. 20 s.	Max. 3 m. Min. 10 s.	Max. 6 cm. Min. 2.5 cm.	Terminated in tetany
		1	109 m.	..	.....	..	..	.....	.....	.....	Fluctuations in tonus
5/26/35	4 hr., 45 m.*	..	.....	1	29 m.	II	31	Max. 45 s. Min. 20 s.	Max. 1 m., 30 s. Min. 10 s.	Max. 4 cm. Min. 2 cm.	Terminated in tetany
		1	16 m.	1	120 m.	II	77	Max. 1 m. Min. 30 s.	Max. 4 m., 15 s. Min. 20 s.	Max. 4 cm. Min. 1.5 cm.	
		1	14 m.	1	70 m.	II	53	Max. 1 m. Min. 35 s.	Max. 2 m. Min. 40 s.	Max. 5 cm. Min. 2.5 cm.	
		1	14 m.	1	22 m.	II	17	Max. 45 s. Min. 20 s.	Max. 1 m., 20 s. Min. 55 s.	Max. 4 cm. Min. 2 cm.	
5/28/35	4 hr., 50 m.	..	.....	1	11 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	7 m.	1	282 m.	II	300	Max. 1 m., 5 s. Min. 45 s.	Max. 1 m., 20 s. Min. 15 s.	Max. 3 cm. Min. 1.5 cm.	Symptoms of biliary colic
5/29/35	5 hr.	..	.....	1	17 m.	II	19	Max. 55 s. Min. 40 s.	Max. 40 s. Min. 5 s.	Max. 5.5 cm. Min. 2 cm.	
		1	14 m.	1	121 m.	II	58	Max. 55 s. Min. 35 s.	Max. 3 m. Min. 25 s.	Max. 5.5 cm. Min. 2.5 cm.	
		1	24 m.	1	74 m.	II	58	Max. 55 s. Min. 30 s.	Max. 5 m. Min. 10 s.	Max. 5.5 cm. Min. 2.5 cm.	
		1	50 m.								
5/30/35	4 hr.	Continuous gastric motility throughout the observation period; symptoms and signs characteristic of biliary colic									

* In this and the following tables m. indicates minutes; s., seconds; Max., maximum, and Min., minimum.

An increase in the usual amplitude of contractions was also observed. Frequently this was as much as from 8 to 10 cm. (table 1). The interval between contractions was about two minutes, and the usual duration of each contraction was about one minute.

The duration of the period of gastric quiescence was variable and was attended by no epigastric distress. During such periods it was possible to observe low grade fluctuations in gastric tonus. Respiratory excursions were evident on the kymographic tracings. With the resumption of intense gastric motility there frequently ensued a recurrence of epigastric distress simultaneous with the severe contractions. In a few instances tracings were obtained during attacks of abdominal pain, which from the standpoint of intensity and radiation was char-

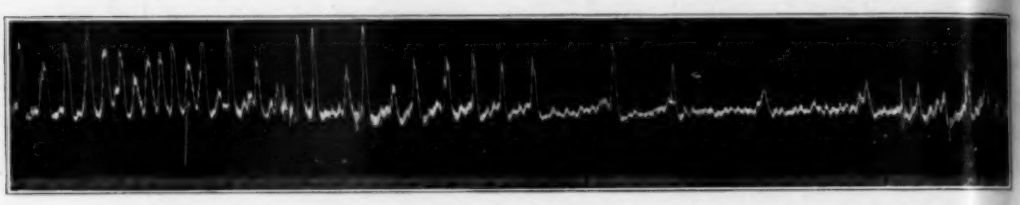

Fig. 1.—Tracing made on May 26, 1935, showing the persistence of gastric motility for one hundred and twenty minutes in a patient with untreated cholelithiasis.

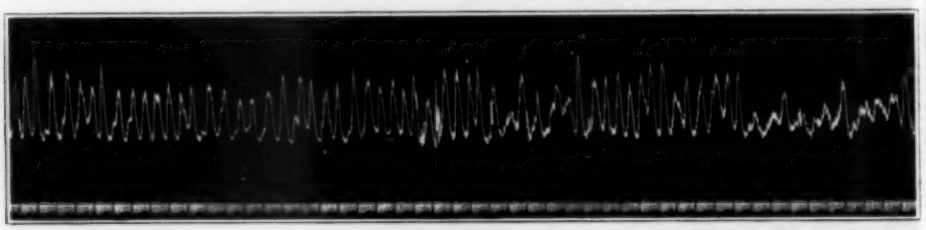

Fig. 2.—Tracing made on May 28, 1935, showing continuous gastric motility during biliary colic. The duration of this period was two hundred and eighty-two minutes.

acteristic of biliary colic. The gastric motility under such circumstances was continuous throughout observation periods of from four to five hours (fig. 2), during which severe contractions were occasionally noted.

The use of general anesthesia prohibited the study of gastric motility during cholecystectomy. However, on the morning of the fourth postoperative day studies were resumed and serial observations continued. These revealed definite hypermotility, more pronounced than that of the control period and persisting throughout an observation period of approximately three hours (fig. 3). The amplitude of the contractions was definitely increased (table 2).

Epigastric distress appeared to parallel these periods of hypermotility during which severe contractions were evident. At the termination of the observations the patients complained of abdominal discomfort, which was invariably interpreted as "gas pains" (fig. 3). Subjectively these sensations persisted for about a week after cholecystectomy. However, the hypermotility persisted for a longer period (table 2). As a late manifestation, the patients complained of intense hunger (fig. 4). The entire duration of the period of postoperative hypermotility was approximately three weeks. Subsequently the motility returned to normal.

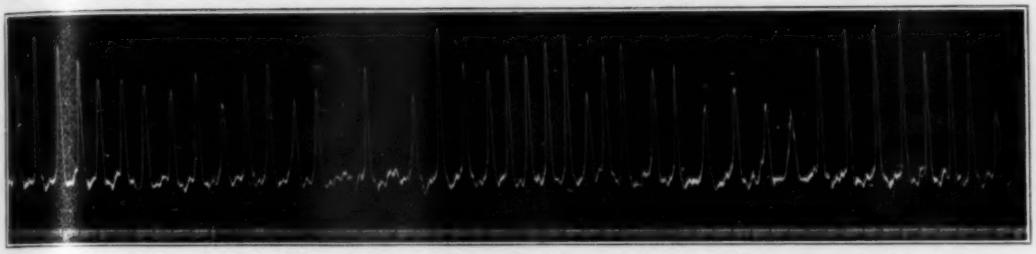

Fig. 3.—Tracing made on June 4, 1935, four days after cholecystectomy, showing intense gastric motility persisting for one hundred and eighty-two minutes. At the termination of this period, the patient complained of "gas pains."

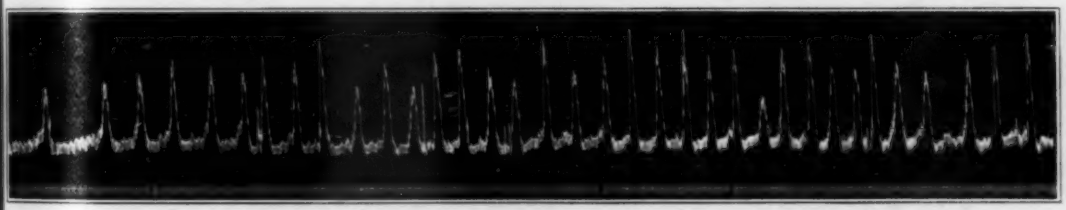

Fig. 4.—Tracing made on June 12, 1935, twelve days after cholecystectomy, showing intense gastric motility associated with hunger. The duration of this period was one hundred and forty-four minutes.

Herniorrhaphy.—Gastric motility was studied on five patients with inguinal hernia. Herniorrhaphy was eventually performed after adequate control studies (table 3). Tracings were also obtained on the morning of operation. On four patients records were made during the operative procedure, which was conducted with low spinal anesthesia. Studies were then resumed immediately after the repair. The duration of these periods of observation varied, but on the whole they were approximately four hours. Daily observations were then made in the morning until the patient was discharged from the hospital. The results were similar in all five patients.

TABLE 2.—*Analysis of Experiments on Gastric Motility After Cholecystectomy*

Date of Experiment	Duration of Experiment	Periods of Quiescence		Periods of Activity		Contractions					Comment
		No.	Duration	No.	Duration	Type	No.	Duration	Interval Between	Amplitude	
6/ 4/35	4 hr.	..	.....	1	182 m.	II	132	Max. 1 m. Min. 25 s.	Max. 3 m., 10 s. Min. 55 s.	Max. 6 cm. Min. 2.5 cm.	
		1	13 m.	1	45 m.	II	27	Max. 50 s. Min. 20 s.	Max. 3 m., 20 s. Min. 55 s.	Max. 6 cm. Min. 3.5 cm.	Complained of gas pains
6/ 5/35	4 hr., 35 m.	..	.....	1	104 m.	II	60	Max. 55 s. Min. 25 s.	Max. 5 m., 40 s. Min. 10 s.	Max. 6.5 cm. Min. 2 cm.	
		1	25 m.	1	75 m.	II	38	Max. 1 m. Min. 30 s.	Max. 4 m. Min. 35 s.	Max. 6.5 cm. Min. 3 cm.	
		1	16 m.	1	55 m.	II	20	Max. 50 s. Min. 20 s.	Max. 4 m., 15 s. Min. 1 m., 30 s.	Max. 6 cm. Min. 2.5 cm.	Complained of gas pains
6/ 6/35	4 hr., 50 m.	..	.....	1	44 m.	II	35	Max. 1 m. Min. 15 s.	Max. 5 m., 20 s. Min. 20 s.	Max. 4.5 cm. Min. 2.5 cm.	
		1	22 m.	1	169 m.	II	121	Max. 55 s. Min. 35 s.	Max. 6 m. Min. 5 s.	Max. 6 cm. Min. 2.5 cm.	
		1	10 m.	1	45 m.	II	38	Max. 1 m., 20 s. Min. 30 s.	Max. 2 m., 15 s. Min. 40 s.	Max. 6 cm. Min. 2 cm.	Complained of gas pains
6/ 8/35	4 hr., 15 m.	..	.....	1	50 m.	II	20	Max. 1 m. Min. 30 s.	Max. 5 m., 10 s. Min. 1 m.	Max. 5 cm. Min. 1.5 cm.	
		1	54 m.	1	20 m.	II	10	Max. 1 m. Min. 40 s.	Max. 3 m. Min. 35 s.	Max. 4.5 cm. Min. 1.5 cm.	
		1 1	12 m. 103 m.	1	16 m.	II	10	Max. 50 s. Min. 30 s.	Max. 2 m., 35 s. Min. 25 s.	Max. 4.5 cm. Min. 2 cm.	
6/10/35	2 hr., 30 m.	Continuous motility throughout observation period; patient complained of nausea and epigastric distress									
6/12/35	4 hr.	..	.....	1	19 m.	II	23	Max. 1 m. Min. 20 s.	Max. 1 m. Min. 10 s.	Max. 4.5 cm. Min. 2.5 cm.	
		1	9 m.	1	144 m.	II	114	Max. 1 m. Min. 40 s.	Max. 2 m., 30 s. Min. 40 s.	Max. 5 cm. Min. 2.5 cm.	

		1	9 m.	1	59 m.	II	36	Max. 45 s. Min. 20 s.	Max. 4 m. Min. 35 s.	Max. 4.5 cm. Min. 1.5 cm.	Complained of hunger
6/13/35	3 hr.	..	.....	1	13 m.	II	16	Max. 1 m. Min. 30 s.	Max. 1 m. Min. 5 s.	Max. 4 cm. Min. 2 cm.	
		1	9 m.	1	158 m.	II	92	Max. 1 m. Min. 45 s.	Max. 5 m. Min. 20 s.	Max. 5 cm. Min. 1.5 cm.	
6/15/35	3 hr.	..	.....	1	7 m.	III	Inestimable	Inestimable	Inestimable	.....	Gastric tetany
		1	5 m.	1	28 m.	II	25	Max. 50 s. Min. 10 s.	Max. 1 m. Min. 15 s.	Max. 4.5 cm. Min. 2 cm.	Terminated in tetany
		1	7 m.	1	133 m.	II	136	Max. 1 m. Min. 25 s.	Max. 1 m., 50 s. Min. 20 s.	Max. 5 cm. Min. 1.5 cm.	Complained of hunger
6/17/35	3 hr., 25 m.	..	.....	1	9 m.	II	10	Max. 25 s. Min. 15 s.	Max. 20 s. Min. 5 s.	Max. 5 cm. Min. 2 cm.	
		1	5 m.	1	39 m.	II	42	Max. 50 s. Min. 20 s.	Max. 1 m. Min. 5 s.	Max. 4 cm. Min. 1.5 cm.	
		1	5 m.	1	147 m.	II	151	Max. 1 m. Min. 25 s.	Max. 1 m., 10 s. Min. 5 s.	Max. 4 cm. Min. 1.5 cm.	
6/18/35	3 hr., 5 m.	..	.....	1	35 m.	II	22	Max. 35 s. Min. 25 s.	Max. 2 m. Min. 10 s.	Max. 5 cm. Min. 2 cm.	
		1	87 m.	1	63 m.	II	16	Max. 45 s. Min. 30 s.	Max. 3 m. Min. 1 m.	Max. 5.5 cm. Min. 2.5 cm.	Complained of hunger
6/19/35	3 hr.,	1	88 m.	1	92 m.	II	32	Max. 40 s. Min. 10 s.	Max. 10 m. Min. 15 s.	Max. 5 cm. Min. 2 cm.	
6/20/35	4 hr., 35 m.	..	.....	1	12 m.	II	9	Max. 1 m., 25 s. Min. 20 s.	Max. 1 m. Min. 10 s.	Max. 5 cm. Min. 3.5 cm.	
		1	99 m.	1	35 m.	II	23	Max. 45 s. Min. 20 s.	Max. 2 m., 40 s. Min. 20 s.	Max. 2.5 cm. Min. 2 cm.	
		1	75 m.	1	54 m.	II	17	Max. 45 s. Min. 10 s.	Max. 2 m., 45 s. Min. 1 m., 30 s.	Max. 3 cm. Min. 1.5 cm.	

TABLE 3.—*Analysis of Experiments on Gastric Motility During the Control Period in a Case of Right Indirect Inguinal Hernia*

Date of Experiment	Duration of Experiment	Periods of Quiescence		Periods of Activity		Contractions					Comment
		No.	Duration	No.	Duration	Type	No.	Duration	Interval Between	Amplitude	
11/ 9/35	4 hr., 40 m.	..	.....	1	43 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	62 m.	1	36 m.	II	34	Max. 1 m., 5 s. Min. 20 s.	Max. 1 m., 20 s. Min. 5 s.	Max. 4 cm. Min. 2 cm.	
		1	48 m.	1	19 m.	II, III	Inestimable	Inestimable	Inestimable	Max. 4 cm. Min. 1.5 cm.	
		1	55 m.	1	17 m.	II	12	Max. 1 m. Min. 30 s.	Max. 1 m., 40 s. Min. 20 s.	Max. 3 cm. Min. 2 cm.	
11/11/35	4 hr.	..	.....	1	62 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 4 cm. Min. 1.5 cm.	
		1	62 m.	1	35 m.	II	35	Max. 1 m. Min. 15 s.	Max. 2 m. Min. 10 s.	Max. 4.5 cm. Min. 2 cm.	
		1	47 m.	1	22 m.	II	27	Max. 30 s. Min. 5 s.	Max. 1 m. Min. 5 s.	Max. 4 cm. Min. 1.5 cm.	Terminated in tetany
		1	12 m.								
11/12/35	4 hr., 30 m.	..	.....	1	25 m.	II	18	Max. 30 s. Min. 10 s.	Max. 2 m. Min. 10 s.	Max. 4.5 cm. Min. 2 cm.	
		1	213 m.	1	35 m.	II	31	Max. 50 s. Min. 5 s.	Max. 1 m. Min. 5 s.	Max. 5 cm. Min. 2 cm.	
11/13/35	5 hr., 45 m.	..	.....	1	23 m.	I	Inestimable	Inestimable	Inestimable	Max. 3 cm. Min. 2 cm.	
		1	59 m.	A few low grade contractions noted							
		1	51 m.	1	38 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 4 cm. Min. 1.5 cm.	
		1	41 m.	1	54 m.	II	40	Max. 1 m. Min. 5 s.	Max. 2 m., 30 s. Min. 5 s.	Max. 5 cm. Min. 2 cm.	
		1	79 m.	..	.....	..	..	.....	.....	.....	Fluctuations in tonus

11/14/35	5 hr., 45 m.	..	.....	1	48 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 4 cm. Min. 1.5 cm.	
		1	27 m.	1	26 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 2.5 cm. Min. 1.5 cm.	
		1	99 m.	1	41 m.	II	33	Max. 1 m., 30 s. Min. 5 s.	Max. 2 m. Min. 5 s.	Max. 4 cm. Min. 2 cm.	
		1	104 m.	Few contractions during this period							
11/15/35	5 hr., 30 m.	..	.....	1	41 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 4.5 cm. Min. 2 cm.	
		1	41 m.	1	34 m.	II	33	Max. 1 m. Min. 5 s.	Max. 2 m., 30 s. Min. 5 s.	Max. 4 cm. Min. 1.5 cm.	Terminated in tetany
		1	76 m.	1	26 m.	II	25	Max. 50 s. Min. 5 s.	Max. 1 m. Min. 5 s.	Max. 4 cm. Min. 2 cm.	
		1	59 m.	1	32 m.	II	30	Max. 55 s. Min. 5 s.	Max. 1 m., 15 s. Min. 5 s.	Max. 5 cm. Min. 2.5 cm.	
		1	21 m.								
11/17/35	4 hr., 35 m.	..	.....	1	34 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	
		1	76 m.	1	35 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	
		1	54 m.	1	23 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 4 cm. Min. 2 cm.	
		1	55 m.								
11/18/35	5 hr., 50 m.	..	.....	1	19 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	Terminated in tetany
		1	49 m.	1	18 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1 cm.	Terminated in tetany
		1	61 m.	1	46 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 3 cm. Min. 1 cm.	
		1	23 m.	1	30 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 4 cm. Min. 1.5 cm.	Terminated in tetany
		1	76 m.	1	28 m.	I, II	Inesti-mable	Inestimable	Inestimable	Max. 4.5 cm. Min. 2 cm.	
11/19/35	3 hr., 15 m.	..	.....	1	26 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 4.5 cm. Min.2.5 cm.	Epigastric distress
		1	105 m.	1	21 m.	II, III	Inesti-mable	Inestimable	Inestimable	Max. 3 cm. Min. 2 cm.	Terminated in tetany
		1	43 m.	Herniorrhaphy on right side at 10:15 a. m.; gastric inhibition throughout operation							

TABLE 4.—*Analysis of Experiments on Gastric Motility During and After Herniorrhaphy on Right Side*

Date of Experiment	Duration of Experiment	Periods of Quiescence		Periods of Activity		Contractions					Comment
		No.	Duration	No.	Duration	Type	No.	Duration	Interval Between	Amplitude	
11/19/35	3 hr., 30 m.	Gastric inhibition throughout observation period; tracing made immediately after herniorrhaphy									
11/20/35	3 hr., 5 m.	Continuous low grade motility; occasional contraction of high amplitude									
11/21/35	3 hr., 15 m.	Continuous low grade motility throughout observation period									
11/22/35	5 hr., 8 m.	..	.....	1	13 m.	II	11	Max. 45 s. Min. 10 s.	Max. 1 m. Min. 20 s.	Max. 3.5 cm. Min. 1.5 cm.	
		1	17 m.	1	18 m.	II	19	Max. 50 s. Min. 5 s.	Max. 1 m., 10 s. Min. 5 s.	Max. 3 cm. Min. 1 cm.	Terminated in tetany
		1	8 m.	1	45 m.	II	43	Max. 1 m. Min. 5 s.	Max. 1 m. Min. 5 s.	Max. 4.5 cm. Min. 1.5 cm.	Complained of gas pains
		1	28 m.	1	179 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 4.5 cm. Min. 1.5 cm.	Complained of gas pains
11/23/35	5 hr.	..	.....	1	29 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	27 m.	1	23 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	
		1	71 m.	1	34 m.	I	Inestimable	Inestimable	Inestimable	Max. 3 cm. Min. 1 cm.	
		1	30 m.	1	86 m.	II	50	Max. 1 m., 10 s. Min. 20 s.	Max. 4 m. Min. 20 s.	Max. 4 cm. Min. 1.5 cm.	Complained of gas pains
11/24/35	4 hr.	..	.....	1	28 m.	II	27	Max. 1 m., 30 s. Min. 5 s.	Max. 1 m. Min. 5 s.	Max. 3.5 cm. Min. 1 cm.	Terminated in tetany
		1	56 m.	1	51 m.	II	38	Max. 1 m., 15 s. Min. 5 s.	Max. 2 m. Min. 5 s.	Max. 3.5 cm. Min. 1 cm.	
		1	78 m.	1	27 m.	II	20	Max. 2 m. Min. 5 s.	Max. 2 m., 10 s. Min. 5 s.	Max. 3.5 cm. Min. 1.5 cm.	
11/25/35	5 hr., 15 m.	Continuous motility throughout observation period; patient complained of hunger									
11/26/35	4 hr., 20 m.	Continuous motility throughout observation period; patient complained of hunger									

11/27/35	5 hr.	..	.....	1	38 m.	II, III	Inestimable	Inestimable	Inestimable	Max. 3 cm. Min. 1 cm.	
		1	27 m.	1	26 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 5 cm. Min. 1.5 cm.	
		1	38 m.	1	54 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	59 m.	1	58 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1 cm.	
11/29/35	4 hr.	..	.....	1	45 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	63 m.	1	132 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 4.5 cm. Min. 1.5 cm.	Complained of hunger
11/30/35	5 hr., 5 m.	..	.....	1	51 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	32 m.	1	34 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	36 m.	1	152 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 3 cm. Min. 1 cm.	
12/ 2/35	5 hr., 25 m.	..	.....	1	28 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	48 m.	1	24 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	
		1	50 m.	1	32 m.	I, II	Inestimable	Inestimable	Inestimable	Max. 3.5 cm. Min. 1.5 cm.	
		1	53 m.	1	41 m.	II	32	Max. 1 m., 10 s. Min. 5 s.	Max. 2 m., 20 s. Min. 5 s.	Max. 3.5 cm. Min. 1.5 cm.	
		1	49 m.								
12/ 3/35	5 hr.	..	.....	1	98 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	49 m.	1	95 m.	II	54	Max. 1 m., 50 s. Min. 5 s.	Max. 3 m. Min. 5 s.	Max. 4.5 cm. Min. 2 cm.	Complained of hunger
		1	58 m.								
12/ 4/35	4 hr., 15 m.	..	.....	1	41 m.	I	Inestimable	Inestimable	Inestimable	Low grade	
		1	50 m.	1	86 m.	II	55	Max. 2 m. Min. 5 s.	Max. 3 m., 10 s. Min. 5 s.	Max. 4.5 cm. Min. 1.5 cm.	Complained of hunger
		1	78 m								

During the control period some variation was noted (table 3). However, the motility was essentially normal (fig. 5 *A*). Immediately preceding hernial repair there appeared to be a definite increase in the amplitude of the contractions, although in many instances the total duration of the periods of activity was relatively short (fig. 5 *B*). There was evidence of considerable associated emotional disturbance. The patients complained of epigastric distress. There was an apparent relationship between the emotional state and the hypermotility. When spinal anesthesia was induced, the stomach became quiescent. This inhibition of gastric motility, associated with a marked reduction in tonus, continued throughout the operation (fig. 5 *C*).

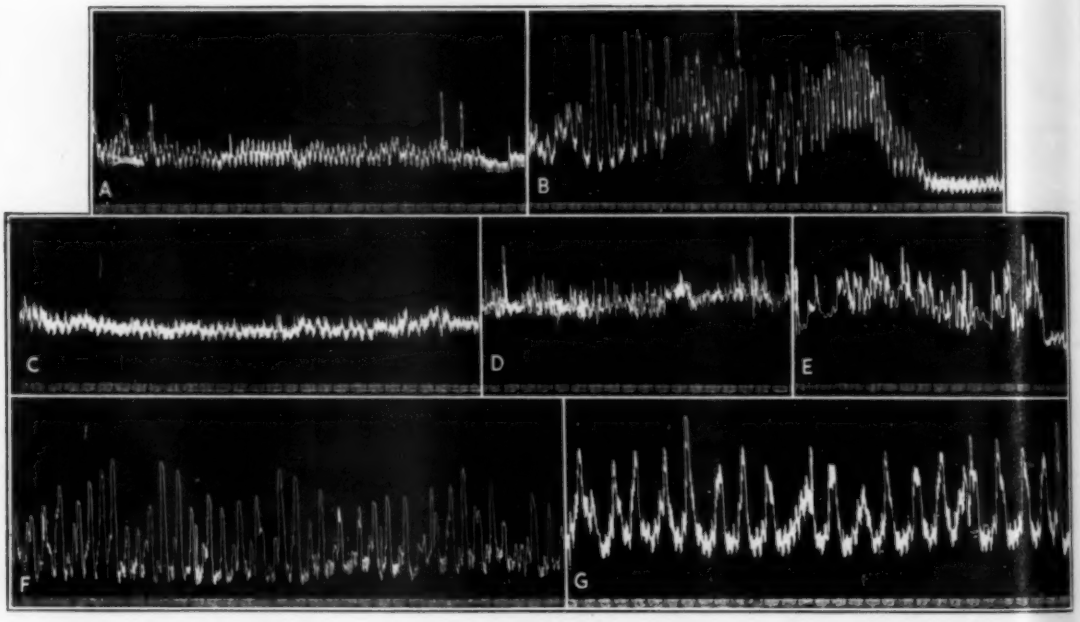

Fig. 5.—Sections *A* to *G* are segments of kymographic tracings obtained on the same patient. *A*, control tracing made on November 12, showing low grade gastric motility in a patient with right indirect inguinal hernia. *B*, tracing made on November 19, about an hour preceding herniorrhaphy. The gastric motility was intense but of short duration. *C*, tracing made on November 19, showing inhibition of the gastric motility during herniorrhaphy. *D*, tracing made on November 19, showing the persistence of inhibition for several hours subsequent to herniorrhaphy. *E*, tracing made on November 20, showing the return of gastric motility twenty-four hours subsequent to herniorrhaphy. *F*, tracing made on November 22, showing continuous motility with intense contractions three days after herniorrhaphy. The duration of this period was one hundred and seventy-nine minutes. At the termination of this observation the patient complained of "gas pains." *G*, tracing made on December 3, two weeks after herniorrhaphy, showing continuous gastric activity associated with hunger.

Inhibition of gastric motility persisted from twenty-four to approximately thirty-six hours subsequent to herniorrhaphy (fig. 5 *D;* table 4). In some instances it continued for seventy-two hours. After this period there was a gradual return of motility (fig. 5 *E*). This manifested itself first by an increase in tonus, superimposed on which were feeble contractions, which grew progressively more intense. About twenty-four to forty-eight hours after the onset of motility, definite hypermotility became evident (fig. 5 *F*). Simultaneous with this the patients complained of epigastric distress, interpreted by them as "gas pains." Hypermotility with intense contractions persisted for about two weeks. However, the sensation of "gas pains" was experienced for approximately one week after operation. Subsequently the patients complained of intense hunger as a late manifestation of hypermotility (fig. 5 *G*). Throughout the postoperative period medication was purposely avoided so that the results of laparotomy would not be obscured. In some patients the hypermotility persisted throughout their entire stay in the hospital. In others the motility approached the preoperative normal prior to discharge.

COMMENT

Preliminary observations on gastric motility revealed some variation among the patients. Therefore, a sufficient number of control studies was made in order to permit of adequate interpretations. The results from each patient were relatively uniform and essentially normal. They were in agreement with the results described by other investigators.[2]

Some evidence of hypermotility during the preoperative period is revealed in the two patients with cholecystitis. Although this was not constant, it was noted with sufficient frequency to be significant. Many tracings exhibited gastric contractions of abnormally great amplitude. When this occurred, the patients frequently complained of abdominal discomfort referable to the epigastrium.

The sensory mechanism which registers abdominal pain in disease of the gallbladder in the conscious patient is exceedingly complex. Theories describing the nervous pathways over which the sensory stimuli travel have been reported in detail elsewhere [3] and therefore are intentionally omitted from this report. The abdominal pain to which we refer was always localized in the epigastrium and was interpreted as a dull, aching, boring distress. It was never associated with nausea or with vomiting. At no time did this discomfort reach sufficient

2. Carlson, A. J.: The Control of Hunger in Health and Disease, Chicago, University of Chicago Press, 1916.

3. Zollinger, Robert: Significance of Pain and Vomiting in Cholelithiasis, J. A. M. A. **105**:1647 (Nov. 23) 1935.

magnitude to indicate termination of the usual period of observation. It did not present the intensity or distribution characteristic of biliary colic.

The frequent occurrence of gastric hypermotility with its associated epigastric distress during the period of cholecystitis with cholelithiasis in which treatment was not given would suggest a relationship between disease of the biliary tract and reflex disturbances in gastric motor function. Carlson [2] made observations on the gastric motility of a patient with disease of the gallbladder. He, too, found that epigastric distress ran parallel with the strong contractions of the empty stomach. He also noted that the contractions were stronger than usual for the age of the patient.

This experimental evidence further indicates that in disease of the biliary tract there may be reflex stimulation of the gastric motor mechanism, with ensuing hypermotility of the stomach. Since epigastric distress accompanied the intense gastric contractions, it would seem that there also ensues a reflex stimulation of the sensory nerves of the stomach. However, the evidence is as yet too indefinite to permit the conclusion that gastric hypermotility is the sole cause of the abdominal distress concurrent with disease of the biliary tract. Doubtless other undetermined extragastric factors play a rôle. Nevertheless, the frequency with which hypermotility of the stomach was associated with epigastric distress in these two patients would indicate concomitant disturbances in reflex gastric motor and sensory functions. Carlson [2] noted that the epigastric pain often appeared greater than warranted by the strength of the gastric contractions. He expressed the opinion that infection of the gallbladder might lead to hyperexcitability of the sensory nervous mechanism of the stomach and duodenum, possibly from the spread of bacterial toxins along the branches of the vagus nerves.

Barber [4] and Barber and Stewart [5] also found hypermotility and pylorospasm associated with disease of the gallbladder. They reported that irritation of the gallbladder, duodenum and appendix increased the rate and force of the gastric contractions. Although the exact cause for this is not clear, it was thought to be of vagal origin.

In a few instances we obtained tracings of the gastric motility during attacks of biliary colic. These revealed continuous motility, frequently persisting throughout the period of observation. The amplitude of these contractions was about from 4 to 5 cm.

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4. Barber, W. Howard: Gas and the Motility of the Surgical Stomach, Ann. Surg. **69**:271, 1919.

5. Barber, W. Howard, and Stewart, George D.: Further Observations upon Reflex Gastric Hypermotility, Proc. Soc. Exper. Biol. & Med. **17**:155, 1919-1920.

Various manifestations of abnormal gastric motor function have been described as associated with disease of the biliary tract; the most common observations have been pylorospasm and hypermotility of the stomach. McCrea [6] reported that irritation and inflammation of the appendix or of the gallbladder may produce pylorospasm, gastric hypermotility and hyperchlorhydria. He even suggested that these may precede the development of ulcer. Smith and Miller [7] demonstrated that irritation of the mucous membrane of the gallbladder of the anesthetized dog with croton oil was followed by a striking increase in the activity of the stomach. After the administration of atropine, there was a decrease in motility. Held and Roemer [8] showed that disease of the gallbladder was likely to produce spasm of the pylorus with active peristalsis of the proximal portion of the stomach. They cited an instance in which gastrospasm was due to gallstones.

The mechanism responsible for the pylorospasm and gastric hypermotility is not clear. However, Hughson [9] found that trauma to the abdominal viscera was capable of producing reflex contractions of the pylorus and gastric hypermotility. He also observed that as a result of peritoneal irritation there ensued an increase in the emptying time of the stomach which persisted for two weeks to a month. He advanced the hypothesis [9b] that spasm of the pylorus resulting from disease either within or adjacent to the abdominal cavity is solely a result of irritation of the peritoneal surfaces.

Contrary to these findings, Ivy [10] reported that mild stimulation of the biliary tract inhibited gastric motility and that distention of the biliary passages caused pylorospasm and also a marked delay in the evacuation of a barium sulfate meal. He noted that a sudden marked distention produced pylorospasm with nausea and vomiting. This was interpreted as being responsible for the dyspeptic symptoms of disease of the gallbladder. In his opinion the pain of biliary colic is not due

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6. McCrea, E. D'Arcy: The Nerves of the Stomach and Their Relation to Surgery, Brit. J. Surg. **13**:621, 1926.

7. Smith, Fred M., and Miller, G. H.: A Study of the Reflex Influence of the Colon, Appendix and Gall Bladder on the Stomach, Am. J. Physiol. **90**:518, 1929.

8. Held, I. W., and Roemer, J.: Gastrospasm: A Clinical and Roentgenological Study, Am. J. M. Sc. **164**:188, 1922.

9. Hughson, Walter: (*a*) The Effect of Vagus Neurotomy on the Pyloric Sphincter: An Experimental Study, J. A. M. A. **88**:1072 (April 2) 1927; (*b*) Reflex Spasm of the Pylorus and Its Relation to Diseases of the Digestive Organs, Arch. Surg. **11**:136 (July) 1925.

10. Ivy, A. C.: (*a*) The Applied Physiology of the Gastro-Intestinal Innervation, Am. J. Digest. Dis. & Nutrition **1**:845, 1935; (*b*) Physiology of the Gall Bladder, Physiol. Rev. **14**:1, 1934.

to pylorospasm, because the type of referred pain is different, and pain referable to the biliary tract may be obtained in man and in the dog after subtotal gastrectomy.

The reports of various investigators thus indicate that pylorospasm is frequently associated with disease of the gallbladder. Whether this is due to local mechanism or to reflex action by way of the splanchnic or vagus nerves is not clear. However, it is well established that motor and inhibitory fibers enter the pylorus through the vagus and splanchnic nerves and that stimulation of either may result in pylorospasm.

Carlson and Litt [11] found that motor disturbances of the pylorus may be induced not only by local pathologic conditions in the stomach and duodenum or by abnormal states of the central nervous system but by excessive irritation of most, if not all, sensory nerves, particularly those of the abdominal viscera. Clinically, it is known that abnormalities in the gastric motor mechanism may occur during periods of emotional disturbances. Many investigators [12] have reported that under such circumstances gastric motility is inhibited. Similar results have been reported by Ivy,[10a] Carlson [2] and others.[13] On the other hand, there are several investigators who maintain that hypermotility is the most common observation.[14] There is consequently a lack of uniformity in the composite results reported in the literature.

Since it was possible to study gastric motility in patients who were experiencing the emotional disturbance which usually precedes a major operation, kymographic tracings were made for periods of about three hours preceding herniorrhaphy and were continued until the patients were taken to the operating room. It was our experience that gastric hypermotility was the most common observation under these circumstances. In many instances the contractions were severe, although the duration of the periods of gastric activity was not unusually prolonged. By means of the electric key and the signal magnet it was possible to observe that epigastric distress occurred during periods of intense motility.

We cannot conclude from our own findings or from the reports of other investigators that either hypermotility or inhibition necessarily occurs during emotional states. It is possible that either may appear,

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11. Carlson, A. J., and Litt, S.: Studies on the Visceral Nervous System: On the Reflex Control of the Pylorus, Arch. Int. Med. **33**:281 (March) 1924.

12. Cannon, W. B.: The Movements of the Stomach Studied by Means of the Röntgen Rays, Am. J. Physiol. **1**:359, 1898. Todd, T. Wingate: Behavior Patterns of the Alimentary Tract, Baltimore, Williams & Wilkins Company, 1930.

13. Luckhardt, Arno B.: The Effect of Dreaming on the Gastric Hunger Contractions, Am. J. Physiol. **39**:330, 1915-1916.

14. Barber.[4] Hughson.[9b]

depending on the preexisting tonus of the stomach and the type of emotional distress manifested. This problem is complex and doubtless is influenced by a multiplicity of factors, which are neither constant nor uniform throughout the studies made by various investigators—hence the discrepancies.

Although it was our experience to observe hypermotility during the emotional changes preceding operation, it is impossible to conclude that it will uniformly occur until emotions are more clearly defined and more accurately investigated.

Motor inhibition of the gastro-intestinal tract is frequently encountered during abdominal laparotomy. This phenomenon was described by Pal as early as 1890.[15] Since then it has been reported by many investigators. Bayliss and Starling[16] found that when the abdomen of a dog was opened in a warm bath of saline solution the intestines were collapsed and motionless. Section of the splanchnic nerves or even radical destruction of the spinal cord was necessary to institute intestinal motility. Similarly, Meltzer and Auer[17] reported that merely cutting the skin in the linea alba of a rabbit was sufficient to abolish peristalsis of the cecum. Auer[18] observed that opening the abdominal cavity of the rabbit caused cessation of gastric movements. Our studies on the human stomach are in agreement with these experimental observations.

We observed inhibition of the gastric motility during herniorrhaphy. This persisted for a varying period, of from twenty-four to approximately seventy-two hours, postoperatively. It would seem that this temporary gastric paresis was due to reflex stimulation of the splanchnic nerves and due to a mechanism similar to that described by Markowitz and Campbell[19] and Ochsner, Gage and Cutting[20] for the production of physiologic ileus. Our tracings show that the motility eventually returned and that hypermotility subsequently ensued.

During this postoperative period of temporary inhibition of gastric motility or physiologic paralysis of the stomach, gastric distention was most evident. It is possible that this distention, if sufficiently extensive,

15. Pal, J.: Ueber den Einfluss des Bauchschnittes auf die Darmbewegung, Centralbl. f. Physiol. **4**:338, 1890.

16. Bayliss, W. M., and Starling, E. H.: The Movements and Innervation of the Small Intestine, J. Physiol. **24**:99, 1899.

17. Meltzer, S. J., and Auer, J.: Peristaltic Movements of the Rabbit's Cecum and Their Inhibition, Proc. Soc. Exper. Biol. & Med. **4**:37, 1907.

18. Auer, John: Gastric Peristalsis Under Normal and Certain Experimental Conditions, Proc. Soc. Exper. Biol. & Med. **4**:8, 1907.

19. Markowitz, J., and Campbell, Walter R.: The Relief of Experimental Ileus by Spinal Anesthesia, Am. J. Physiol. **81**:101, 1927.

20. Ochsner, Alton; Gage, I. M., and Cutting, R. A.: Treatment of Ileus by Splanchnic Anesthesia, J. A. M. A. **90**:1847 (June 9) 1928.

acts as a stimulus to the gastric motor mechanism so that hypermotility follows. In 1904 May [21] reported that hypermotility occurred when the dog's excised stomach was inflated with air. Lim, Ivy and McCarthy [22] made similar observations. They found that when overdistention occurred there ensued a decrease in motility and in tone. It would seem from our observations that the distention occurring during this physiologic adynamic period increased until it reached the maximal threshold level peculiar to each patient. The ensuing hypermotility was then probably the result of mechanical stimulation to the gastric motor mechanism in a manner similar to that previously reported. This postoperative hypermotility persisted for from ten days to two weeks. Occasionally it was seen throughout the entire period of postoperative convalescence. In other instances it returned to normal prior to the patient's discharge from the hospital.

It is probable that reflex stimulation of the gastric sensory mechanism occurs simultaneously with the hypermotility of the stomach. The most common subjective sensation experienced by the patients during the early postoperative period of increased gastric activity was a form of epigastric distress interpreted by them as gas pains. These were intermittent and coincided with the severe contractions. Subsequently the patients complained of intense hunger as a late manifestation of increased gastric activity.

Although the early postoperative epigastric distress was interpreted as gas pains and although this occurred during a period of intense gastric activity, we do not think that the hypermotility alone is responsible for this sensation. Obviously there are other factors intimately involved in this problem. Nevertheless, there was sufficient evidence to indicate that in the genesis of postoperative epigastric distress the gastric motor mechanism assumed an important rôle. We would think that inhibition of the gastric motility followed by the hypermotility has certain clinical implications which are significant in understanding the nature of early postoperative disturbances.

The spinal anesthesia which was used during the repair of the hernias did not extend beyond the operative field. The work of other investigators [23] reveals that hypermotility of the gastro-intestinal tract ensues only if the spinal anesthesia produces paralysis of the splanchnic nerves. We observed no evidence that this occurred during our studies.

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21. May, W. Page: The Innervation of the Sphincters and Musculature of the Stomach, J. Physiol. **31**:260, 1904.

22. Lim, Robert K. S.; Ivy, A. C., and McCarthy, J. E.: Contributions to the Physiology of Gastric Secretion: I. Gastric Secretion by Local (Mechanical and Chemical) Stimulation, Quart. J. Exper. Physiol. **15**:13, 1925.

23. Markowitz and Campbell.[19] Ochsner, Gage and Cutting.[20]

Although we cannot outline with certainty the physiologic mechanism responsible for the production of this inhibition during laparotomy and for a varying period subsequently, we do not think that spinal anesthesia per se is the cause. The work of Markowitz and Campbell [19] and Ochsner, Gage and Cutting [20] would lead us to believe that spinal anesthesia is responsible for hypermotility as a result of paralysis of the splanchnic nerves. This hypermotility occurred after an extremely short latent period.

The hypermotility evident on our tracings appeared from one to three days subsequent to operation. Therefore, it is our belief that the mechanism which is responsible for the inhibition of gastric motility which we observed during operation and for a varying period subsequently is similar to the mechanism which is responsible for the inhibition of the intestinal motility during laparotomy, a phenomenon already described.[19] The hypermotility which followed might possibly be due to stimulation of the gastric motor mechanism by the mechanical distention of the stomach occurring during the period of inhibition.

CONCLUSIONS

Gastric motility was studied by the balloon and kymograph method on seven patients on whom laparotomy was performed. The results of this investigation revealed the following facts:

1. There was a tendency toward hypermotility of the stomach in patients with untreated cholecystitis associated with cholelithiasis.

2. Continuous gastric motility was associated with occasional severe contractions during biliary colic.

3. Increased gastric hypermotility persisted for from two to three weeks after cholecystectomy. During the first week of this increased motility the patients experienced epigastric distress interpreted by them as gas pain; subsequently they complained of intense hunger as a late manifestation of hypermotility.

4. Normal gastric motility was present in five patients with inguinal hernia until immediately preceding hernial repair, at which time short but intense periods of gastric activity, associated with emotional disturbance, were observed.

5. Inhibition of gastric motility persisted throughout herniorrhaphy and for from twenty-four to seventy-two hours subsequently.

6. Gastric hypermotility subsequent to inhibition persisted for from two to three weeks, during which the subjective sensations were similar to those described after cholecystectomy.

CARCINOMA OF THE FEMALE BREAST

WITH SPECIAL CONSIDERATION OF PREOPERATIVE IRRADIATION: A PRELIMINARY REPORT

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In the recent surgical literature there are seven articles to which I should like to call special attention.[1] Six of these deal with preoperative irradiation in the treatment of carcinoma of the breast. The seventh is concerned chiefly with the statistical study of cancers of the breast treated by surgical measures alone and by surgical measures and postoperative irradiation. From one of these articles, namely, that of Adair and Stewart, I have taken the following data on eighty-one cases of operable carcinoma of the breast in which preoperative irradiation was followed by a radical Halstead operation.

Radium element pack	39 cases
No residual cancer	11 cases (28%)
Roentgen therapy, 200 kilovolts	42 cases
No residual cancer	7 cases (16%)

In each of these cases biopsy was performed preliminary to irradiation.

In the *Nova Scotia Medical Bulletin* of June 1935 Bloodgood gave his conclusions in favor of irradiation preliminary to operation for carcinoma of the breast.

Bloodgood and Stewart, in a brief paper in the *Southern Medical Journal* for July 1936, made further remarks on the preoperative irradiation of cancer of the breast and reported a case which I shall refer to again in this article.

From the clinic of the late Dr. Joseph C. Bloodgood and his associates.

1. Bloodgood, Joseph Colt: The Problem of the Breast Tumor, Nova Scotia M. Bull. **14**:307 (June) 1935. Cohn, L. Clarence: Benign Breast Lesions, with Special Consideration of Border-Line Tumors: Cancer of the Breast and the Newer Conception of Preoperative Irradiation, West Virginia M. J. **31**:1 (Jan.) 1936. Adair, Frank E., and Stewart, Fred W.: The Value of Preoperative Irradiation in Breast Cancer, Ann. Surg. **102**:254 (Aug.) 1935. Hutchinson, R. G.: The Value of Radiation Therapy in the Treatment of Carcinoma of the Breast, Surg., Gynec. & Obst. **62**:653 (April) 1936. Nicolson, W. P., and Berman, M. D.: Carcinoma of the Breast: A Study of Five Year End-Results, Ann. Surg. **103**:683 (May) 1936. Graham, Allen: Cancer of the Breast, with Particular Reference to Irradiation as a Factor in End-Results, Pennsylvania M. J. **39**:561 (May) 1936. Bloodgood, Joseph Colt, and Stewart, G. A.: Preoperative Irradiation of Breast Tumors, South. M. J. **29**:651 (July) 1936.

My first experience with the preoperative irradiation of operable cancer of the breast was in 1927, almost ten years ago, and was what might be described as an accident. This concerned a married woman aged 42, whose youngest child was 9 years of age.

I examined the patient with Dr. Bloodgood on March 28, 1927, and we found a gland the size of a 5 cent piece in the base of the right axilla, with a smaller definite nodule in the periphery of the right breast just adjoining the gland. She had felt the lump only four days. Although the roentgenogram of the chest was normal, we decided to use irradiation for the first treatment because of the possibility of lymphosarcoma. When I examined her on May 2, more than four weeks later, and two weeks after roentgen therapy (200 kilovolts), the lump had decreased in size two-thirds and was smaller than a 10 cent piece. As the nodule did not disappear, I decided to do a complete axillary dissection. I made the usual axillary incision as in the complete operation for cancer of the breast, divided the pectoralis major and minor muscles, exposed the axillary vein and dissected the axilla from above downward. On introducing the finger beneath the palpable tumor, finding that the tumor felt like cancer and that it would be necessary to remove a considerable portion of the breast to give the tumor the proper margin, I decided on the usual complete operation for cancer of the breast, doing the dissection of the wall of the chest after the axillary dissection—the reverse of our usual method. In doing the axillary dissection I was unable to palpate any glands except this nodule in the base of the axilla, which had been palpated prior to the operation.

The frozen section showed a cellular medullary carcinoma, and there was no metastasis to the axillary glands.

The patient is now (September 1936) well, almost ten years after operation. This tumor must be looked on as carcinoma in the axillary quadrant of the breast or as carcinoma in aberrant breast tissue in the axilla.

On April 28, 1930, more than three years later, a widow 35 years of age consulted Dr. Bloodgood with a microscopic section from a carcinoma excised from the left breast on Dec. 9, 1929, four and a half months previously. The tumor had been present only one month before it was excised, and it was clinically benign. At the examination Dr. Bloodgood was unable to palpate any nodules in the scar, breast or axilla. He decided to give irradiation, which was carried out from April 28 to May 9.

Dr. Bloodgood decided on irradiation first because he felt that the chief danger was that metastasis had already taken place during the four and one-half month interval since the operation. When the patient returned on October 13, five months later, examination of the breast gave entirely negative results, but a second course of irradiation was given over the breast, axilla and mediastinum. There has been no further operation other than the excision of the tumor, and the patient is now well, six and a half years since the operation.

On Dec. 12, 1931, Mrs. T. consulted us with an operable but very advanced carcinoma of the breast in which there was beginning carcinoma *en cuirasse* (fig. 1 *A*). Figure 1 *B* shows the result three months after irradiation, and the patient is now well, five years after treatment. The only treatment was one course of irradiation. No biopsy was done in this case; so the only evidence that this patient had cancer was clinical.

Preoperative irradiation was not taken up seriously in this clinic until the summer of 1932, four years ago, when Dr. Bloodgood returned

from a visit to some of the outstanding radiologic clinics in Europe. At that time I was much opposed to giving a patient with carcinoma of the breast preoperative irradiation and felt that there was a distinct danger in delaying the operation for irradiation, and I believe this is the view of the majority of my colleagues today.

On June 15, 1932, more than four years ago, Mrs. M. J., aged 57, consulted us because of a large, visible mass occupying the entire right breast, producing a dome-shaped bulging of the breast with the nipple at the top of the dome. The mass was adherent to the skin and the nipple, and there was fixation of the nipple and of the areola. In spite of the extensive involvement of the breast, there were no palpable glands in the axilla. The tumor had been present for five months. The patient was referred to the Kelly Hospital for preoperative irradiation. This was carried out by Dr. Edmond Kelly, from whose notes the following information was taken: During June 1932 a total of 8 gram hours of radium treatment was given at a distance of 5 cm. directly to the right axilla with the pack; also

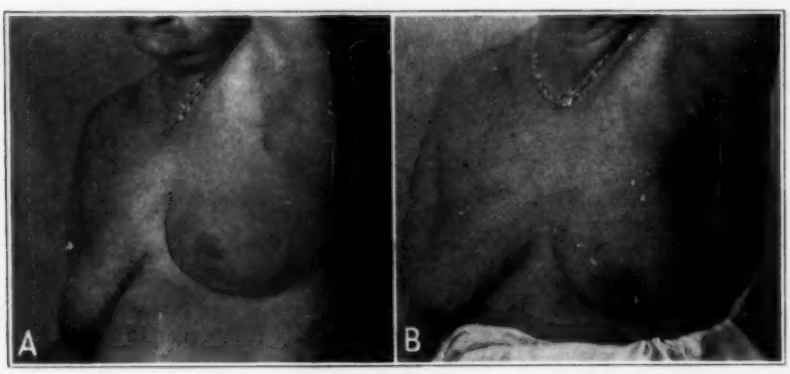

Fig. 1.—*A*, photograph of a patient taken on Dec. 12, 1931, showing an advanced, clinically malignant tumor of the breast. *B*, the same patient on March 23, 1932, after treatment by irradiation only. The patient was still well in May 1936.

5 gram hours at a distance of 4 cm. to the right lateral aspect of the right breast and similar treatment to the left lateral aspect of the right breast. In addition to this, nine 4.2 millicurie gold needles were implanted permanently into the substance of the breast, scattered equally through its periphery, yielding a total radiation of approximately 4.5 gram hours during the life of the needles (twenty-eight days). The patient was then given 5 gram hours at a distance of 4 cm. to the right supraclavicular space. In addition, she was given high voltage roentgen therapy through a portal 10 by 10 cm., 900 roentgens each to the anterior and the posterior surface of the right axilla.

The total amount of radiation was given in ten days and was completed on June 25. On September 30, more than three months later, Dr. Bloodgood and I did the operation, which consisted of an exploratory incision (definite cancer was observed in the frozen sections) and the excision of the breast and pectoral muscles but not of the axilla. The axillary dissection was omitted because of the precarious condition of the patient. Figure 2 shows the appearance of the patient before and after operation, and figure 3, the microscopic pathologic picture. The

patient is now well more than four years after operation and is our first example of a patient with an operable but advanced carcinoma of the breast who was cured for four years by complete irradiation and incomplete surgical excision.

Later I shall give the figures on excision of the breast only (including dissection of the wall of the chest) with and without preoperative irradiation. The total number of patients in this group is small, and the therapy is not, in my opinion, the treatment of choice. The foregoing case marked the beginning of our intensive study of preoperative irradiation for carcinoma of the breast.

DATA ON CASES OF PRIMARY AND RECURRENT CARCINOMA OF THE BREAST

For the purposes of the present study all cases of primary and recurrent carcinoma of the breast observed in the clinic from January

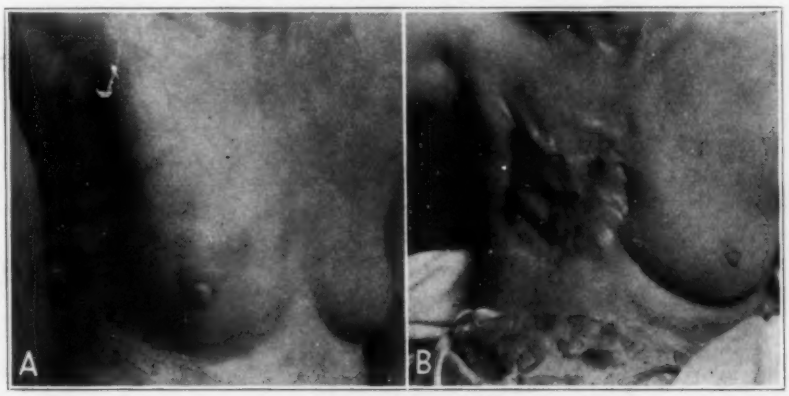

Fig. 2.—*A*, photograph of a patient with a cancer involving the larger part of the breast. *B*, the same patient after operation. (These photographs were previously published by Bloodgood, J. C.: Am. J. Surg. **28**:490, 1935.)

1931 to December 1935 are included, or, in other words, during the immediate preceding five year period, and while it is clear that any report on such a recent series of cases may be criticized as premature, it was felt that a preliminary report at this time might bring out and emphasize what seems to be the keynote of the problem, which is that there is apparently no danger in delaying the complete operation for one course of preoperative irradiation.

A summary of the data to be analyzed is presented in table 1, and while it is realized that the number of patients studied is not large, the numbers in the different groups seem to be near enough equal so as to allow some basis for comparison, and I plan to report on the same series again at the end of five years.

At this writing (September 1936) from four years to nine months has elapsed since therapy in the cases in which irradiation was employed, with one exception, and in the cases in which surgical treatment only was used from two to five years or more has elapsed.

In table 1, the patients in groups 1, 2 and 3 include the majority of those with primary carcinoma, and it is these patients who attract particular attention. When one compares the data in table 2 with those

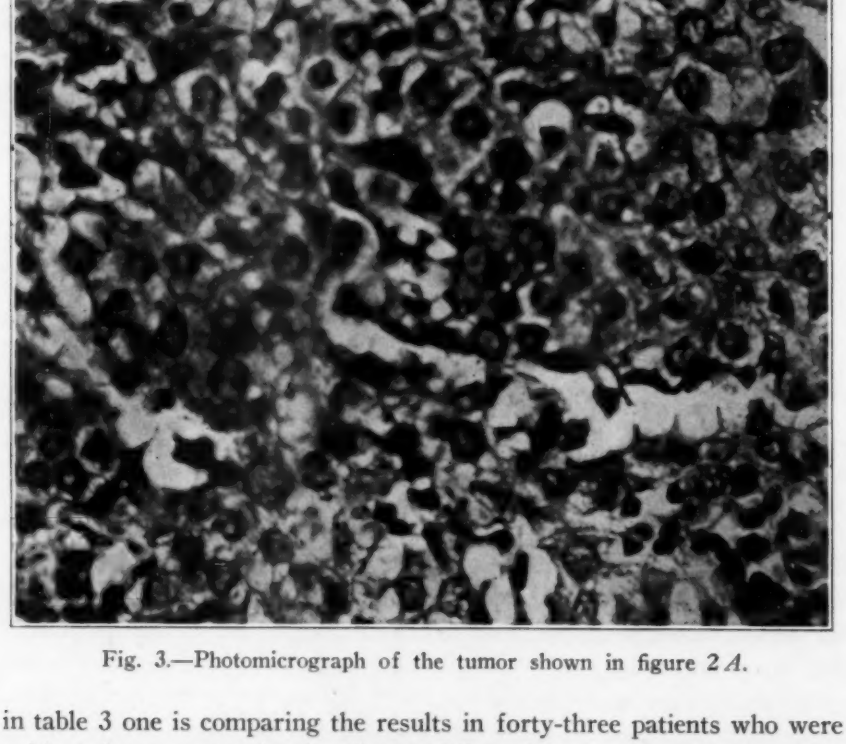

Fig. 3.—Photomicrograph of the tumor shown in figure 2 *A*.

in table 3 one is comparing the results in forty-three patients who were subjected to preoperative irradiation followed by the complete operation with those in fifty-one patients who underwent complete operation without preoperative irradiation. Of the former group, thirty, or 71 per cent, are living and free from recurrence, while of the latter group, only seventeen, or 33 per cent, are living and free from recurrence. While eight of the former group died of cancer and four are living with recur-

rence (28 per cent), twenty-eight of the latter group are dead of cancer and three are living with recurrence (60 per cent).

However, for the group of thirty patients living and free from recurrence after preoperative irradiation followed by the complete operation the average length of time since irradiation was begun is only one year and ten months, whereas for the group of seventeen patients living and free from recurrence after the complete operation only the average length of time since operation is three years and four months. For the group of twelve patients dead or living with recurrence after preoperative irradiation plus the complete operation, the average length of time since irradiation was begun is one year and nine months, and for the group of thirty-one patients dead or living with recurrence after the complete operation only, the average length of time since operation is one year and seven months.

TABLE 1.—*Classification of Patients According to Treatment and Results*

Group	Status	No. of Patients
1	Preoperative irradiation and complete operation	43
2	Complete operation; no preoperative irradiation	51
3	Irradiation only, including excision of tumor and biopsy	42
4	Excision of breast only	9
5	Recurrent carcinoma	41
6	Insufficient record	6
	Total	192

If such limited statistics indicate anything, it is interesting to note that there is apparently no evidence that the addition of preoperative irradiation prolongs the duration of life in those persons who are to die of the disease.

It should again be emphasized at this point that the figures and percentages given in tables 2 and 3 and in the subsequent tables are merely the figures and percentages as they have been worked out for the present study and should not be interpreted except in this light. They are not given as proof or even as arguments in favor of preoperative irradiation or that preoperative irradiation followed by the complete operation will cure, for five years or longer, a larger percentage of patients. The solution of this problem, at least as far as this series of patients is concerned, will have to await the passage of time. On the other hand, the reason for the publication of these figures is that sufficient time seems to have elapsed to indicate that there is apparently no danger in delaying the complete operation for one course of preoperative irradiation, provided sufficient time elapses between the irradiation and the operation, and in our experience the optimum time for the operation is from two and a half to three months after the irradiation is completed. This interval allows the skin and deeper struc-

tures to return to normal, even though there have been blistering and epitheliitis from the irradiation.

At the beginning we were inclined to operate too soon after completion of the irradiation. The only death occurred in the following case:

The patient consulted us on May 10, 1934, with a section from a tumor which had been removed from her breast six days previously. The section showed medul-

TABLE 2.—*End-Results for Patients in Group 1*

Total number of patients	43	
Living, free from recurrence	30	(71%)
Death from cancer	8	(28%)
Living with recurrence	4	
Death postoperatively, due to infection	1	
Average length of time since irradiation was begun for patients living and free from recurrence	1 yr., 10 mo.	
Average length of time since irradiation was begun for patients dead or living with recurrence	1 yr., 9 mo.	

TABLE 3.—*End-Results for Patients in Group 2*

Total number of patients	51	
Living, free from recurrence	17	(33%)
Death from cancer	28	(60%)
Living with recurrence	3	
No note	2	
Death from another cause	1	
Average length of time since operation for patients living and free from recurrence	3 yr., 4 mo.	
Average duration of life after operation for patients dead or living with recurrence	1 yr., 7 mo.	

TABLE 4.—*End-Results for Patients in Group 3*

Total number of patients	42	
Living and well	13	(31%)
Death from cancer	26	(62%)
Lost from observation	2	(7%)
Death from another cause	1	
Average duration of life from onset of tumor (from history) until death	23 mo.	
Average duration of life from time of admission (irradiation) until death	14 mo.	
Average length of time since admission (irradiation) for patients living and well	2 yr., 3 mo.	
Average length of time since onset of disease for patients living and well	2 yr., 8 mo.	

lary carcinoma, and there was a small gland palpable in the axilla. She was given a course of preoperative irradiation from May 10 to May 26. When she returned on July 10, less than two months later, there was a small nodule in the upper end of the scar of two weeks' duration, and for this reason we decided on a second course of irradiation before operating. The second course was begun on July 10 and was completed on July 20, at which time the little nodule that we had felt in the scar ten days previously had entirely disappeared, and the little gland that we had palpated in the axilla on May 10 was no longer palpable. When the patient returned for her next examination, on October 22, there was no evidence of any

recurrence of the disease in the breast or the axilla. Three months later, at our examination on Jan. 25, 1935. there was another nodule in the scar of one month's duration, and a third course of irradiation was given, from Janury 25 to February 12. On February 15, three days later, I did the complete operation. Sections from the breast and axilla showed no residual cancer. Dr. Gey reported the tissue culture to be negative.

Fig. 4.—High power photomicrograph of a smear made from a biopsy specimen secured by aspiration, showing cells distinctly of the malignant type. (This photograph was previously published by Bloodgood, J. C., and Stewart, G. A.: South. M. J. **29**:651, 1936.)

The wound became infected with hemolytic streptococci, and in spite of our recognizing the infection at once and starting treatment with diluted solution of sodium hypochlorite, the patient died on the tenth day after operation. This is the only infection due to hemolytic streptococci that we have had in a breast wound since the influenza epidemic in 1918, and it is quite possible that it was an accidental occurrence, but I have always felt that the tissues were unable to take care of this infection because they had not yet recovered from the injury of the irradiation.

Of the forty-three patients on whom preoperative irradiation and the complete operation were done, twenty-eight received irradiation without a biopsy because the tumor was clinically malignant, and one patient

TABLE 5.—*Data Concerning Biopsy and Tissue Culture on Patients in Group 1*

Total number of patients		43
Biopsy		16
Before irradiation	15	
Excision of tumor	14	
Aspiration	1	
After irradiation—followed by complete operation	1	
No biopsy		27
Tissue cultures made		23
Positive	6 (29%)	
Negative	15 (71%)	
Questionable	2	
No cultures made		20

TABLE 6.—*Data Concerning Tissue Culture on Patients in Group 2*

Total number of patients	51
Tissue culture positive	2
Tissue culture negative	2
No culture taken	47
Postoperative irradiation as a routine	9
Postoperative irradiation after recurrence	10

was treated with irradiation followed by a biopsy and immediately after the biopsy by the complete operation. Biopsy was done for fifteen patients before irradiation, excision of the tumor being done in fourteen instances and aspiration in one instance (fig. 4). Tissue cultures were taken in twenty-three of the forty-three cases and were reported by Dr. Gey as positive in six instances (29 per cent) and negative in fifteen instances (71 per cent). The tissue culture was questionable in two cases (table 5). What the significance of the growth of the tumor or the lack of the growth of the tumor in tissue culture is as yet is not known because in the fifty-one cases in which the complete operation was done without preoperative irradiation the tissue culture was taken in only four instances. It was positive twice and negative twice (table 6).

In connection with biopsy, there is one case I shall report a little more in detail because the procedure followed was so unorthodox and brings up the question of what shall be done for a clinically benign tumor which in a frozen section proves to be cancer.

On Nov. 5, 1935, I explored a clinically benign tumor, smaller than a 25 cent piece, of two months' duration, in a woman 46 years of age. After excising the tumor and bisecting it, I was convinced that it was carcinoma, and immediate preparation and study of a frozen section confirmed this. The gross and microscopic pictures are presented in figures 5 and 6. In this case, instead of disinfecting the wound and doing the complete operation, I simply closed the wound and referred the patient to Dr. Neill for irradiation, which was started at once to the supraclavicular area and axilla; after about one week, during which time the wound in the breast healed, the irradiation was carried over the breast. Figure 7 is the photograph of the patient after irradiation.

Fig. 5.—Photograph of the gross specimen of a clinically benign tumor.

On Jan. 21, 1936, three months later, I did the complete operation (fig. 8). There was no metastasis to the axillary glands, and there was no residual carcinoma in the breast. A culture of tissue from the tumor was positive, and one was also reported to be positive after the complete operation.

The chances of curing a patient with a tumor like this for five years by surgical measures alone are 70 per cent, and it remains to be seen whether the addition of this preoperative irradiation will increase or decrease the chances of a cure.

Of the forty-three patients subjected to preoperative irradiation and the complete operation, we found no residual cancer in the breast or axilla in ten (24 per cent), and of these ten, biopsy was performed for seven. We found residual cancer in the breast or axilla of thirty-two patients (76 per cent), as shown in table 7. These figures should be compared with those of Adair and Stewart.

The factors which seem to influence the prognosis are the grade of malignancy of the tumor (Broders), the presence or absence of glandular involvement and the extent of the involvement. The grade of the tumor is really an index of the rapidity of the growth, whereas the involvement of the axillary glands is an index of the extent of the growth and depends to some extent on the grade, but perhaps more on the duration of the tumor. I shall take up the question of involvement

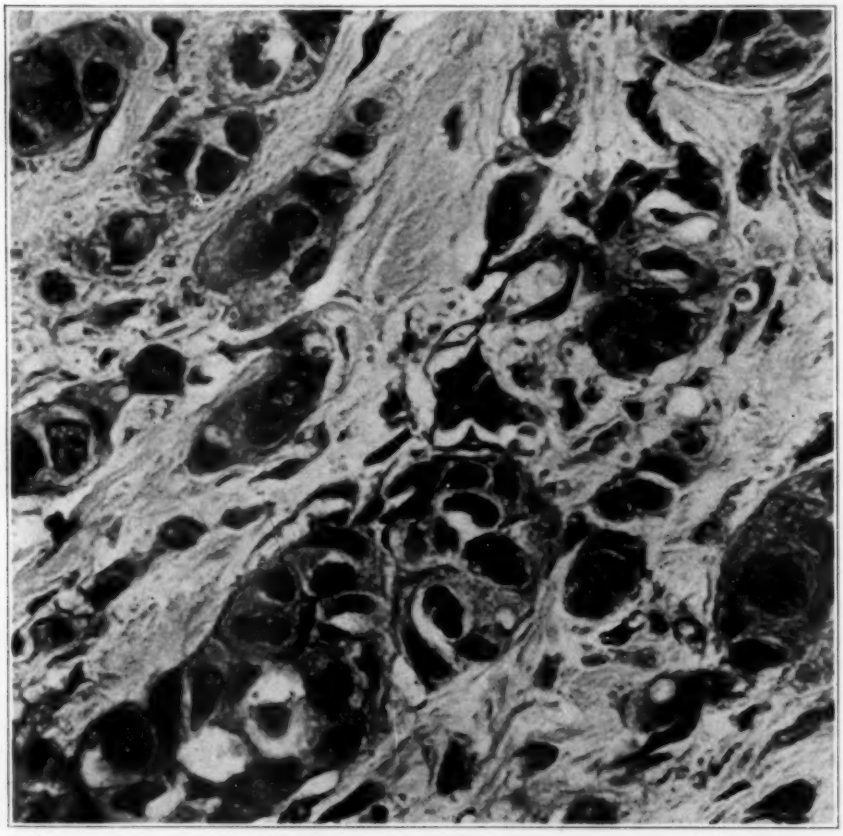

Fig. 6.—Photomicrograph of a clinically benign tumor.

of the glands first (tables 8 and 9). Of the group of forty-three patients on whom preoperative irradiation and the complete operation were done, the glands were involved in eighteen (44 per cent) and showed no metastasis in twenty-three (56 per cent). Of the group with no metastasis to the axillary glands, twenty-one are well (91 per cent), one died of cancer and one died of some other cause. Of the eighteen patients in whom the glands were involved, eight (44 per cent) are well and ten (56 per cent) died of cancer (table 8).

Of the group of fifty-one patients subjected to the complete operation but without preoperative irradiation, there was no metastasis to the axillary glands in fourteen (30 per cent).

Fig. 7.—Photograph of a patient after excision of the tumor and irradiation. For the gross and microscopic appearance of the tumor see figures 5 and 6.

Fig. 8.—Photograph of the patient shown in figure 7 after complete operation.

Frequently I have been able to palpate axillary glands prior to irradiation and three months later prior to operation all evidence of

palpable axillary glands had disappeared; it seems quite possible that the irradiation is responsible for the higher percentage of patients free from axillary involvement—56 per cent in the group treated by irradiation as compared with 30 per cent in the group on whom irradiation was not done. From tables 8 and 9, it is seen that 91 per cent of the patients who were free from involvement of the glands were well after preoperative irradiation and the complete operation and that 90 per cent of those without metastasis to the glands were well after complete

TABLE 7.—*Data Concerning Residual Cancer in Patients in Group 1*

Total number of patients		43	
No residual cancer in breast or axilla		10	(24%)
Biopsy, consisting of excision of tumor	7		
No biopsy	2		
Sections unsatisfactory	1		
Residual cancer in breast or axilla		32	(76%)
No note		1	

TABLE 8.—*End-Results in Relation to Metastasis in Patients in Group 1*

Total number of patients		43	
No metastases to glands		23	(56%)
Well	21 (91%)		
Death from cancer	1		
Death from another cause	1		
Metastases to glands		18	(44%)
Well	8 (44%)[1]		
Death from cancer	10 (56%)		
No note		2	

TABLE 9.—*End-Results in Relation to Metastasis in Patients in Group 2*

Total number of patients		51	
No metastasis to glands		14	(30%)
Well	9 (90%)		
Death from cancer	1 (10%)		
Death from another cause	1		
No record	3		
Metastasis to glands		33	(70%)
Well	8 (24%)		
Death from cancer	25 (76%)		
No note		4	

operation only; so there is no evidence yet that irradiation is of value in early cases of cancer in which there is no involvement of the axillary glands, but there is no evidence that it is harmful, even though it may be unnecessary. On the other hand, 44 per cent of the patients with metastasis to the glands who were subjected to preoperative irradiation plus complete operation were well, as compared with 24 per cent of those who were treated with complete operation alone. It is to be emphasized, however, that the patients who received irradiation on the average have been treated more recently than those who were not treated by irradiation.

When only the glands at the base of the axilla are involved, the complete operation alone offers about 25 per cent chances of a five year cure. In the cases included in this report, which I have already stated is not a report of five year cures, there were seven patients on whom the complete operation without preliminary irradiation was done in whom only the glands of the base of the axilla showed metastasis, and of these seven only two are well (29 per cent) and five are dead of cancer (71 per cent). In the group on whom preoperative irradiation plus the complete operation was done, there are six in whom only the

TABLE 10.—*End-Results in Relation to Extent of Metastasis in Patients in Group 1*

Total number of patients		43
Glands plus		18 (44%)
Well	8 (44%)	
Death from cancer	10 (56%)	
Gland 1 plus		6
Well	4 (66.66%)	
Death from cancer	2 (33.33%)	
Glands 1, 2 plus		1 well
Glands 1, 2, 3 plus		8
Well	1 (12.5%)	
Death from cancer	7 (88.5%)	

TABLE 11.—*End-Results in Relation to Extent of Metastasis in Patients in Group 2*

Total number of patients		51
Glands plus		33 (70%)
Well	8 (24%)	
Death from cancer	25 (76%)	
Glands 1 plus		7
Well	2 (29%)	
Dead	5 (71%)	
Glands 1, 2 plus		6
Well	1 (17%)	
Death from cancer	5 (83%)	
Glands 1, 2, 3 plus		12
Well	2 (17%)	
Death from cancer	10 (83%)	

glands of the base of the axilla showed metastasis, and of these, four (66.6 per cent) are well. The actual number of patients with involvement of the glands of the base and the middle of the axilla or of the base, the middle and the highest apex of the axilla is greater in the group who did not receive irradiation, but the number of patients in each group is too small to allow any conclusions (tables 10 and 11).

Tables 12 and 13 are compiled to show the ultimate result according to the microscopic grading of the tumor in the forty-three patients on whom preoperative irradiation and the complete operation were done and in the fifty-one on whom the complete operation was performed but no preoperative irradiation was given. Of the patients in the first group, 75 per cent with a tumor of grade 1 or grade 2 are well and

25 per cent died of cancer, whereas of those in the second group, 70 per cent are well and 30 per cent died of cancer, an insignificant difference in the percentages. Sixty-one per cent of the patients with tumors of grade 3 who were given irradiation are well, as against 23 per cent of the patients with this grade of tumor who were not given irradiation. The number of patients with a tumor of grade 4 in both groups is too small to be of any value in this study. From these figures it would seem that if there is any value in preoperative irradiation it is for the tumor of grade 3.

TABLE 12.—*End-Results in Patients in Group 1 in Relation to Grade of Tumor*

Total number of patients		43
Grade 1 tumor		48
Well	6 (75%)	
Death from cancer	2 (25%)	
Grade 2 tumor		12
Well	9 (75%)	
Death from cancer	3 (25%)	
Grade 3 tumor		18
Well	11 (61%)	
Death from cancer	7 (39%)	
Grade 4 tumor		2
Well	1	
Death from another cause	1	
No residual cancer		2
Sections unsatisfactory		1

TABLE 13.—*End-Results in Patients in Group 2 in Relation to Grade of Tumor*

Total number of patients		51
Grade 1 tumor		None
Grade 2 tumor		13
Well	7 (70%)	
Death from cancer	3 (30%)	
Death from another cause	1	
No note	2	
Grade 3 tumor		27
Well	6 (23%)	
Death from cancer	20 (77%)	
No note	1	
Grade 4 tumor		2
Well	1 (50%)	
Death from cancer	1 (50%)	
No note		9

In this series of one hundred and ninety-two patients it has already been noted that there were forty-two with primary carcinoma on whom irradiation only or excision of the tumor or some other type of biopsy plus irradiation was performed (table 4). Up to the present time irradiation has not been advised as the only treatment in any case of primary operable carcinoma of the breast except to a few patients over 70 years of age in whom the tumor was arrested by irradiation and to two younger women with acute carcinoma, and in these all evidence of the tumor in the breast and axillary glands disappeared rapidly under irradiation.

Of this group of forty-two patients, thirteen had clinically operable tumors. Two of these were the young women with acute carcinomas, and the remaining eleven were older women. There were a few patients in this group who refused the complete operation. Of the thirteen with operable tumors who were given irradiation only, 66 per cent are well (table 14). As the tumor and axillary glands disappeared so rapidly under irradiation in the two patients with acute carcinoma, we decided not to operate because we felt that the chief danger was distant metastases and that should the tumor recur in the breast or axillary glands we could always operate. These patients were kept under constant observation. One of the patients died of cancer one year and eight months after the beginning of irradiation, but there was no recurrence in the

TABLE 14.—*End-Results in Patients in Group 3*

Total number of patients		42
No biopsy		29
Biopsy		13
Excision of tumor	8	
Clinically operable tumors		13
Well	8 (66%)	
Average duration of time since admission (irradiation)		2 yr., 3 mo.
Death from cancer	4 (33%)	
Average duration of life after irradiation		1 yr., 8 mo.

TABLE 15.—*End-Results in Patients Subjected to Excision of the Breast Only*

Total number of patients		9
No recurrence		4
Average duration of time since operation	3 yr., 3 mo.	
Death from cancer		4
Average duration of life since operation	2 yr.	
Death from other cause		1
Duration of life after operation (no recurrence)	2 yr., 11 mo.	

breast or axilla until two months before death, at which time she was bedridden because of metastases to the pelvis, lower lumbar vertebrae and chest. The other patient is well and free from clinical evidence of cancer one year and two months after irradiation began, during which period there have been two courses of irradiation.

The other twenty-nine patients who received irradiation only had clinically inoperable tumors, so there was no choice in treatment other than irradiation. It has already been noted in table 4 that 31 per cent of these patients are well and that the average period of time since irradiation was begun is two years and three months.

One of the reasons for publishing this material at this time as a preliminary report is that there is such a great difference of opinion between radiologists of experience and surgeons of experience. Some radiologists feel that carcinoma of the breast is no longer a surgical problem and that the treatment should be left entirely to the radiolo-

gists. They strongly object to the complete operation but occasionally agree to a simple mastectomy. On the other hand, the majority of surgeons of experience have not accepted as yet preoperative irradiation and feel that the best results at present can be obtained by the complete operation or by the complete operation and postoperative irradiation.

My associates and I have had a very limited experience in this clinic with excision of the breast only and have taken the point of view that it is an illogical operation in most instances because it is too much of an operation for a benign lesion and not enough of an operation for cancer. In this series there are nine patients who were subjected to excision of the breast only or excision of the breast and pectoral muscles but not the axillary dissection. Of these nine, four are well, four died of cancer and one died of another cause. The average length of time after operation in the four patients who are well is two years and eleven months (table 15). Of these nine patients, four received preoperative irradiation, and three are well (tables 16 and 17). The

TABLE 16.—*Results in Patients Subjected to Preoperative Irradiation Followed by Excision of the Breast*

Total number of patients	4	
Well, no recurrence	3	(75%)
Average duration of time since irradiation	3 yr., 5 mo.	
Death from cancer	1	
Duration of life after irradiation	5 mo.	
Duration of life after operation	2 mo.	

average length of time since irradiation was begun is three years and five months. There were five patients in the group of nine who received no preoperative irradiation (tables 17 and 18). One patient is well two years and seven months after operation, three died of cancer (average duration of life since irradiation, three years) and one died of another cause in two years and eleven months without any clinical evidence of recurrence of metastases.

From these figures alone, which of course are limited, there is no actual proof that preoperative irradiation followed by simple excision of the breast is not as good as preoperative irradiation followed by the complete operation, yet if the reader will refer again to table 8 he will find that 44 per cent of the patients who had preoperative irradiation followed by the complete operation had demonstrable metastases in the axillary glands, and personally I feel very strongly that there should be no restriction of the complete operation because of preoperative irradiation.

The surgeon will have one opportunity only to cure a patient with carcinoma of the breast. When a clinically benign tumor proves in a frozen section to be cancer or is suggestive of cancer, he has the choice of doing the complete operation at once or closing the wound, giving

preoperative irradiation and after the proper interval of time doing the complete operation. In the doubtful cases, while the irradiation is going on the section can be submitted to other pathologists for their opinions. When the sections show cancer, even though all palpable evidence of disease was completely removed for biopsy, for the present anyway, I still feel that the complete operation should follow.

TABLE 17.—*Data on Patients Subjected to Excision of Breast Only in Regard to Grade of Tumor and Tissue Culture*

Total number of patients		9
Number of patients subjected to preoperative irradiation		4
Well (tumors, grade 1, 2, 2)	3	
Death from cancer (grade 3)	1	
No preoperative irradiation		5
Well (tumor, grade 3)	1	
Death from cancer (grade 2, 3 and no tissue from 1)	3	
Lost from observation (no tissue)	1	
Tissue culture		2
Negative (preoperative irradiation)	1	
Inaccurate (preoperative irradiation)	1	

TABLE 18.—*End-Results in Patients Subjected to Excision of Breast Without Preoperative Irradiation*

Total number of patients	5	
Well, no recurrence	1	(25%)
Duration of time since operation: 2 yr., 7 mo.		
Death from cancer	3	(75%)
Average duration of life after operation: 3 yr.		
Death from other cause	1	
Duration of life after operation: 2 yr., 11 mo.		

TABLE 19.—*End-Results for Patients in Group 5*

Total number of patients	41	
Well	4	(9%)
Average duration of time since admission to clinic: 1 yr.		
Death from cancer	36	(88%)
Average duration of life after admission to clinic: 1 yr., 4 mo.		
Death from another cause (suicide)	1	(2%)

The chances of curing a recurrent carcinoma of the breast are so small that I have about decided no longer to advise operation. Of the forty-one patients with recurrent carcinoma in this series, only four (9 per cent) are well, and the average length of time since these four were admitted to the clinic is only one year (table 19). Some of these recurrent carcinomas will be operable and present an apparently favorable prognosis, but the fact alone that they are recurrent almost excludes any possibility of a cure by surgical measures or by preoperative irradiation and surgical treatment. Perhaps even in this group it may be wise to treat by irradiation only.

GASTRIC SURGERY AND GASTROSCOPY

DIFFERENTIAL DIAGNOSIS OF BENIGN AND MALIGNANT LESIONS; OPERABILITY OF TUMORS AS DETERMINED BY GASTROSCOPY; EARLY DIAGNOSIS OF GASTRIC CARCINOMA; THE POSTOPERATIVE STOMACH

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The purpose of this paper is to show the usefulness and value of gastroscopy to the surgeon.[1] The following questions will be discussed: 1. Is a lesion of the stomach, which has been diagnosed by other methods, a benign ulcer or a carcinoma? 2. Is cancer of the stomach operable or not? 3. Of what value is gastroscopy in the early diagnosis of carcinoma, and may it improve the operative results? 4. What are the reasons for the abdominal distress which so frequently follows operations for gastric lesions?

We shall not include the results of the numerous gastroscopies carried out by one of us (R. S.) in Germany, because the records cannot be controlled. Thus we shall restrict ourselves to the gastroscopic examinations carried out at the Billings Hospital of the University of Chicago Clinics.[2] The majority of the patients were referred for gastroscopy by Dr. W. L. Palmer, chief of the gastro-intestinal department. A total of four hundred and sixty-five gastroscopies were performed; forty-one of the patients on whom gastroscopy was per-

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All surgical work was done by the Surgical Department of the University of Chicago Clinics unless otherwise stipulated. Splendid cooperation was given by the Department of Radiology (Dr. Hodges).

1. Schindler, R.: Lehrbuch und Atlas der Gastroskopie, Munich, J. F. Lehmanns, 1923; Diagnostic Gastroscopy, with Special Reference to the Flexible Gastroscope, J. A. M. A. **105**:352-355 (Aug. 3) 1935; A New Tube for Anaesthetization of the Hypopharynx, Am. J. Digest. Dis. & Nutrition **2**:281-282 (July) 1935; Gastroscopy in Thirty Cases of Gastric Neoplasm, Arch. Int. Med. **32**:635-646 (Oct.) 1923. Moutier, F.: Traité de gastroscopie et de pathologie endoscopique de l'estomac, Paris, Masson & Cie, 1935.

2. The flexible Schindler-Wolf gastroscope was used in all examinations. This instrument may be obtained from the Zeiss Corporation, 485 Fifth Avenue, New York.

formed also received surgical treatment, and it is this group which furnishes the material for our paper. Biopsic control was possible in twenty cases; the remaining twenty-one are presented because of the interesting observations which they offer. Since more than one gastroscopy was done on many of our patients, a total of seventy-eight gastroscopies were performed on our group of forty-one patients. We realize that complete presentations of such a large amount of material tends to make the paper rather detailed, but since so little work has been done relative to the value of gastroscopy in cases involving operations for gastric lesions, we believe that surgeons will appreciate these details as being valuable for purposes of study.

A. GASTRIC ULCER AND CARCINOMA VISUALIZED BY GASTROSCOPY BEFORE OPERATION

DIFFERENTIAL DIAGNOSIS

Benign Ulcer.—The difference between a benign and a malignant pyloric obstruction is characteristic gastroscopically, although it is well known that a duodenal ulcer cannot be seen with the gastroscope. Recent statistics indicate that a prepyloric ulcer is very rare and is generally carcinomatous. We have never seen one gastroscopically, but since the lesser curvature of the antrum is infrequently seen with the gastroscope, this observation is of less importance than is the experience of the pathologist and the surgeon. It is, however, sometimes difficult even for the pathologist to decide, in the gross specimen, whether a pyloric obstruction is benign or malignant. In all instances of carcinomatous obstruction of the pylorus the protruding carcinomatous tissue was seen by gastroscopy, whereas in cases of ulcer of the pyloric channel or of benign pyloric obstruction the pylorus was usually not seen because adhesions draw it backward so as to remove it from the field of vision. It may be mentioned that in cases of uncomplicated duodenal ulcer the pylorus is often seen very well, and its activity shows no deviation from the normal. The following case illustrates the uncertainty of diagnosis in many of these cases as well as the difficulty of deciding definitely the nature of the lesion by examination of the gross specimen.

CASE 1.—*History.*—A white Canadian man aged 38, seen on Aug. 14, 1934, had suffered from epigastric distress for one year without remission. He had lost 35 pounds (15.9 Kg.) in weight and had a sensation of tenderness and fulness which became so acute at times that he felt he would burst.* When he induced vomiting, he obtained relief. The vomitus practically always contained undigested food. At no time were tarry stools noted, and there were no other complaints.

Physical Examination.—There was some tenderness in the epigastrium. A large peristaltic wave was seen which moved across the entire upper two thirds of the abdomen. No definite mass was felt. The other organs were normal.

Laboratory Examination.—One thousand, two hundred cubic centimeters of gastric fluid obtained after an Ewald test meal showed: free acid, 37; total acid, 59; lactic acid, 0, and blood, a trace. Microscopic examination of the contents gave normal results. The reaction of the stools to the benzidine test for blood varied from negative to 3 plus.

Roentgen Examination.—The roentgenogram was very suggestive of carcinoma of the antrum of the stomach, with obstruction, retention and dilatation. The condition was thought to be carcinoma of the antrum, but the possibility of an ulcer could not be ruled out.

Gastroscopy.—The stomach contained considerable fluid, which covered the greater curvature. The antrum was well seen, however, particularly in its greater curvature. The pylorus was not completely visible. The mucous membrane was somewhat moist and swollen, but there was no tumor visible; the musculus sphincter antri was very prominent. The mucosa of the body showed no pathologic changes. The gastroscopic diagnosis was gastritis of the antrum.

Clinical Impression.—On the basis of the clinical picture the condition was thought to be carcinoma of the stomach with pyloric obstruction.

Operation (August 20).—The stomach was found to be greatly enlarged, and a hard inflammatory mass was felt at the pylorus. The pyloric antrum and the first part of the duodenum were resected. The duodenum and the end of the stomach were closed, and a posterior gastro-enterostomy was performed with the first part of the jejunum.

Pathologic Examination.—The base of the ulcer was necrotic and yellow and extended well into the wall of the stomach. There were radiating fibrous strands and adhesions about its outer aspect in which there were palpable lymph nodes indicating carcinomatous invasion. Section of the wall showed it to be thickened and to contain translucent and fibrous material. Microscopic examination of the ulcer showed it to be benign.

The diagnostic course of this case is typical. A definite obstruction of the pylorus gave the clinical impression that a carcinoma was present; roentgen examination also indicated carcinoma, although the roentgenologist stated that a benign ulcer could not definitely be ruled out. The surgeon, however, thought that the lesion which he removed was benign, but on gross examination it was thought to be carcinomatous. Microscopic examination proved that the lesion was a benign ulcer. Since no tumorous tissue was seen at the pylorus on gastroscopic examination, we were safe in concluding that carcinoma was not present but that the obstruction was due to a benign lesion with secondary inflammation of the mucosa of the antrum.

After the operation gastroscopy was done three different times. The results will be discussed in section B.

The following case presents a general agreement concerning the nature of the lesion.

Case 2.—*History.*—A man aged 47, seen on Oct. 9, 1934, stated that he had had complaints referable to the stomach for the past seventeen years with rather frequent vomiting but no pain. The past few years he had suffered from disten-

tion, which had been relieved by sodium bicarbonate. The appetite had remained good, although the patient had lost 25 pounds (11 Kg.) in the past year and a half.

Physical Examination.—Nothing abnormal was found.

Laboratory Examination.—Two hundred and thirty cubic centimeters of gastric fluid obtained after an Ewald test meal showed: free acid, 40; total acid, 94, and blood, 0. The stools gave a 4 plus reaction to the benzidine test for blood.

Roentgen Examination.—The roentgenogram showed a stenosing ulcer of the pyloric channel with a surrounding inflammatory reaction and resulting gastric retention. Early pyloric neoplasm could not be absolutely ruled out.

Gastroscopy.—The entire stomach, including the pylorus, was well seen. There were no pathologic changes present.

Clinical Impression.—On the basis of the clinical picture the condition was thought to be a duodenal ulcer with high grade stenosis.

Operation (December 8).—Operation was delayed because of the indecision of the patient. A large duodenal ulcer was found which produced almost complete pyloric stenosis. The lesion was situated on the posterior wall about 3 cm. distal to the pylorus. A posterior gastro-enterostomy was performed. No pathologic examination was possible. The patient returned to the clinic on Feb. 4, 1935; he had gained 5 Kg. and looked extremely well.

Although this case was evidently one of obstructive duodenal ulcer, a neoplasm could not be ruled out with certainty by roentgen examination, whereas the gastroscopic examination was conclusive, since the pylorus was well seen and no carcinomatous changes were visualized.

There was complete agreement in diagnosis of the following case.

CASE 3.—*History.*—A man aged 67 had suffered from pain in the epigastrium below the xiphoid process for the past twenty-five years, with restlessness, irritability and fatigue becoming prominent during the past two years. There was a loss of weight of 10 pounds (4.5 Kg.) during the past six months.

Physical Examination.—A mass was felt in the epigastrium above the umbilicus that moved with peristaltic waves, which were both visible and palpable.

Roentgen Examination.—The roentgenogram showed high grade pyloric obstruction, thought almost certainly to be the result of a chronic duodenal ulcer.

Gastroscopy.—It was difficult to find the pylorus, but finally the entire antrum and the lower half of the pylorus were well seen. The lower parts of the greater curvature were edematous, as were other parts of the stomach to a lesser degree. In the upper parts of the body red spots were seen. The diagnosis on the basis of the gastroscopic findings was superficial edematous gastritis.

Operation.—The wall of the stomach was found to be markedly thickened. There was definite scarring of the duodenum just beyond the pylorus. A posterior gastro-enterostomy was done, and the patient made an uneventful recovery.

The gastroscopic findings were typical of benign obstruction, especially the high grade edema of the antrum, which is seen only in this condition.

In the differential diagnosis of gastric ulcer other signs are important. It is frequently impossible to differentiate such an ulcer from a carcinoma even in the gross specimen, and one may think that it is just as

Explanation of Plate

A, gastroscopic picture of the penetrating benign ulcer of the lesser curvature of the stomach in case 4. The edge of the crater-like ulcer is sharp, and the cardiac part of its floor is brownish. Blood is shown oozing from the lowest part of the floor and accumulating in the dependent part (upward in the picture). The adjacent mucosa is excessively swollen and inflamed. Compare this illustration with figures 1, 2, 3 and 4.

B, gastroscopic picture of the infiltrating carcinoma with ulceration in the case in which operation was not performed. In the center of the picture there is a dark red ulceration. All the other parts of the stomach visualized with the gastroscope were stiff and infiltrated, and the surface was necrotic. The necrosis extended up to the cardia. Compare this illustration with figure 8. Gastroscopy proved immediately the inoperability of the tumor.

C, gastroscopic picture in case 7, showing ulcerative carcinoma. The ulcer is filled with brown clotted blood. The gray ridge discloses the malignant character of the ulcer.

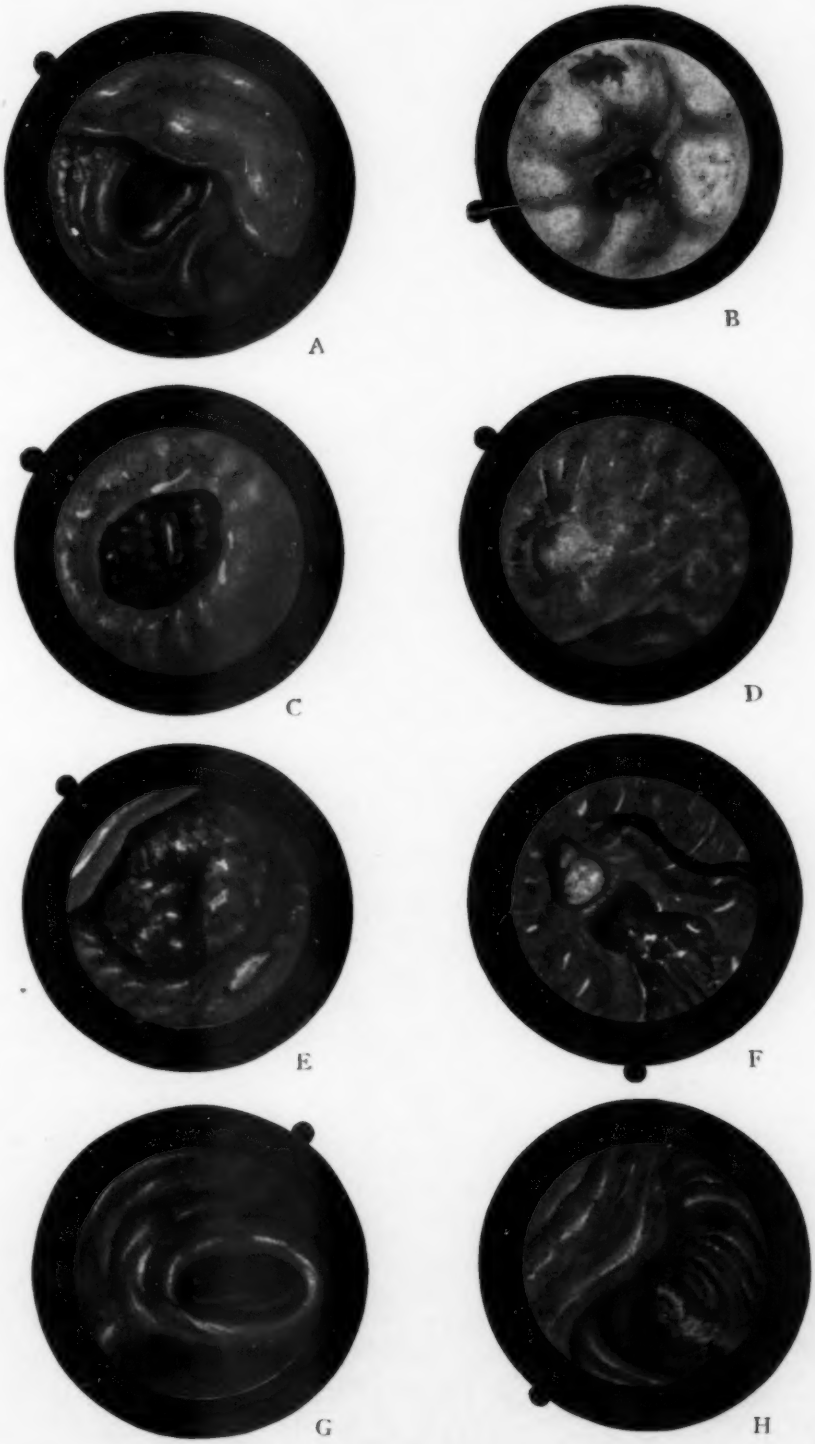
D, gastroscopic picture, also from case 7, made four weeks after *C*. The ulcer as well as the niche (fig. 5) were shallower, but at the pyloric side a nodular infiltration was seen. Compare this picture with figures 6 and 7.

E, gastroscopic picture in case 13, showing an ulcerated carcinoma around the pylorus with a sharply limited wall. The fold above the tumor is the "angulus." The whitish areas are remainders of food. Radical operation was possible. Compare this picture with figures 9 and 10.

F, gastroscopic picture in case 14, showing the patent gastro-enterostomy opening in the center. Intestinal juice flows into the stomach. At the side of the stoma there is a white ulcer larger than the stoma. A silk suture is seen coming out of the mucosa and hanging freely into the cavity of the stomach.

G, gastroscopic picture in case 21, showing the opening phase of the gastro-enterostomy stoma. The objective of the gastroscope is turned toward the anterior wall; the pylorus, which is well seen in figure 12, is not visible. The circular Kerkring folds of the jejunum are seen.

H, gastroscopic picture in case 26, showing a considerable portion of the jejunum in the right lower quadrant. About 2 cm. below the margin a crater-like deep ulcer is seen. The margin is partly covered with pus, and about it mucosal hemorrhages are seen.



PLATE

difficult by gastroscopic examination. This is definitely not so. As a rule these ulcers are seen distinctly gastroscopically, and the circulating blood makes the coloring quite characteristic. It can be further observed that the edge of a carcinoma is never as sharp as it appears to be in the gross specimen after resection and also that the floor of a carcinomatous ulcer blends gradually with the surrounding mucous membrane. If the edge is entirely sharp the lesion may be diagnosed as a benign ulcer; if the edge is not so sharp and the floor is ragged, dirty and irregular, it may safely be called a malignant ulcer. The sharpness of the wall is a more important differential point than is the abnormality of the floor of the lesion.

CASE 4.—*History.*—A man aged 51, seen on Oct. 24, 1934, about five years previously had an attack of epigastric distress which was thought to be the result of a peptic ulcer. He was placed on the Sippy diet, and the pains disappeared. About one month before he vomited and felt very weak. He placed himself on a milk diet, during which time his distress continued, and his emesis occurred at least twice daily. He had lost 24 pounds (10.9 Kg.) in weight during the past two months.

Physical Examination.—Moderate tenderness was felt in the epigastrium. All other findings were normal.

Laboratory Examination.—One hundred and sixty-five cubic centimeters of gastric fluid obtained after an Ewald test meal showed: free acid, 35, and total acid, 51. Microscopic examination of the contents gave normal results. The stools gave a 4 plus reaction to the benzidine test for blood.

Roentgen Examination (October 26).—There was an enormous penetrating ulcer at the lesser curvature of the stomach; the film showed the thickened gastric rugae of the opposite wall. The size of the lesion and the mucosal changes indicated, but did not prove, the possibility that the ulcer might be carcinomatous (fig. 1).

First Gastroscopy (October 30).—On the lesser curvature of the stomach a large ulcer was seen just above the angulus. It was very deep, and its edges were sharp and undermined. The color of that part of the floor which lay toward the cardia was reddish brown, whereas that near the pylorus was whitish gray. In this region of the ulcer fresh light red blood was oozing from the base and flowed toward the cardiac side of the floor; the mucous membrane of the stomach in this region was definitely spotted. Slightly nearer the cardia the mucous membrane of the anterior wall appeared swollen; it did not reflect light and appeared to be divided into sections by small creases. The entire lower parts of the anterior wall also presented these changes and further disclosed one rather large hemorrhage. On the other hand, all portions of the greater curvature of the posterior wall and of the upper parts of the anterior wall were normal. On the basis of the gastroscopic findings a diagnosis was made of (1) deep bleeding benign ulcer of the lesser curvature of the body above the angulus and (2) marked hypertrophic hemorrhagic gastritis of the anterior wall. (See *A* in the color plate.)

Second Gastroscopy (November 19).—The ulcer was instantly seen. It had lost much of its depth but was still large. Its edge was quite sharp, and the floor was dirty gray. Whitish membranes passed from the mucous membrane of

the cardiac side of the ulcer to its floor. The antrum and pylorus were normal. The hypertrophic gastritis of the anterior wall was as distinctly seen as at the last examination and showed no improvement. The other parts of the stomach were normal (fig. 2).

Fig. 1 (case 4).—Roentgenographic picture of the stomach. Because of the size of the niche, the diagnosis of a malignant growth could not be ruled out. Compare this picture with the gastroscopic picture in *A* of the color plate. Gastroscopically, the benign nature of the lesion was evident.

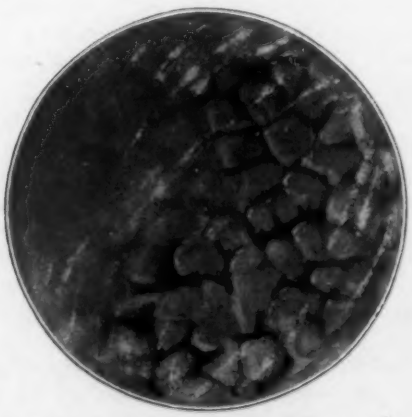


Fig. 2 (case 4).—Severe hypertrophic gastritis in the vicinity of the ulcer, as visualized gastroscopically. The mucous membrane is swollen and nodular ("cobblestone formation"), and hemorrhage is seen (see left side of illustration). (From Schindler, R.: Am. J. Digest. Dis. & Nutrition **2**:655 [Jan.] 1936.)

Second Roentgen Examination (November 26).—There was no diminution in the size of the large penetrating ulcer of the lesser curvature of the stomach.

Clinical Impression.—Considering the history of symptoms of long standing, the ulcer was believed to be benign. The patient was advised to have an operation.

Operation (November 30).—A large ulcer was found situated high on the lesser curvature. Its crater was large enough to admit the tip of a thumb. Gastric resection was done, with uneventful recovery.

Pathologic Examination.—About 5 cm. above the pylorus there was a deep penetrating ulcer, measuring 2.75 by 2 cm., with a sharply defined border and a punched-out appearance. The edges were slightly undermined. The base consisted of adhesive omental tissue. The rugae which radiated from the ulcer were much flattened for an area 8 cm. in diameter around the lesion. This region appeared hyperemic, swollen and hypertrophic (fig. 3). The border of the ulcer was raised and indurated but was not stony hard. The rest of the mucosa appeared practically normal except for petechiae. Section through the edge of the crater of the ulcer showed an ulcer penetrating through all walls of the stomach; the floor was flat and composed of fibrosed omentum, whereas the sides

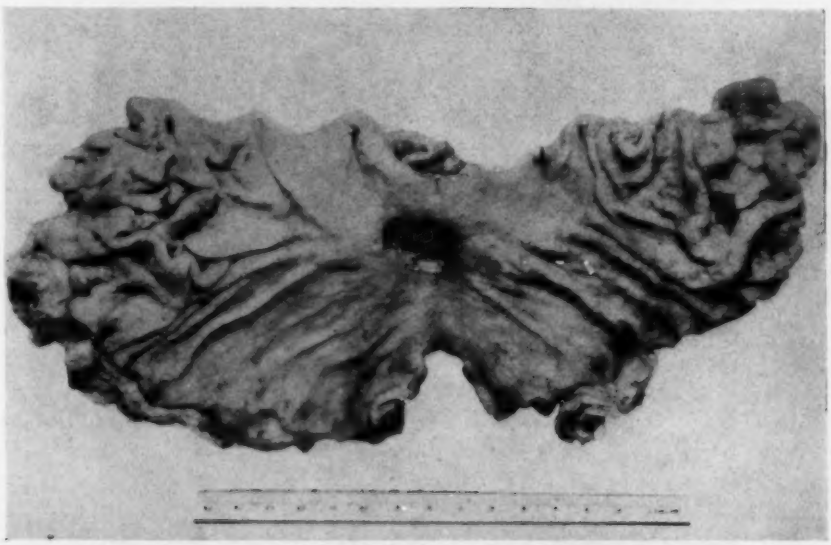


Fig. 3 (case 4).—Resected gastric ulcer. Compare this illustration with figure 1 and with *A* in the colored plate. The gastritic nodules seen gastroscopically can be observed in the gross specimen.

were steep and abrupt. Round cell and eosinophilic infiltration was seen involving the fibrosed omentum and the wall of the stomach. Sections from various other parts of the stomach showed varying degrees of eosinophilia and polymorphonuclear invasion but no evidence of ulceration or malignancy (fig. 4).

In this case the clinical picture and the gastroscopic picture were in complete agreement. It should be noted, however, that the roentgenologist considered the possibility of a malignant ulcer more likely, whereas the gastroscopic picture definitely showed the ulcer to be benign. Another point of interest is that the ulcer appeared shallower on the second gastroscopic examination, whereas there was no difference in the results of the two roentgen examinations. It cannot be proved, of

course, which impression was correct. The microscopic specimen confirmed the gastroscopic findings of severe hypertrophic gastritis around the ulcer.

Because of the fact that so many of the deep ulcers which were observed gastroscopically healed under the medical management of

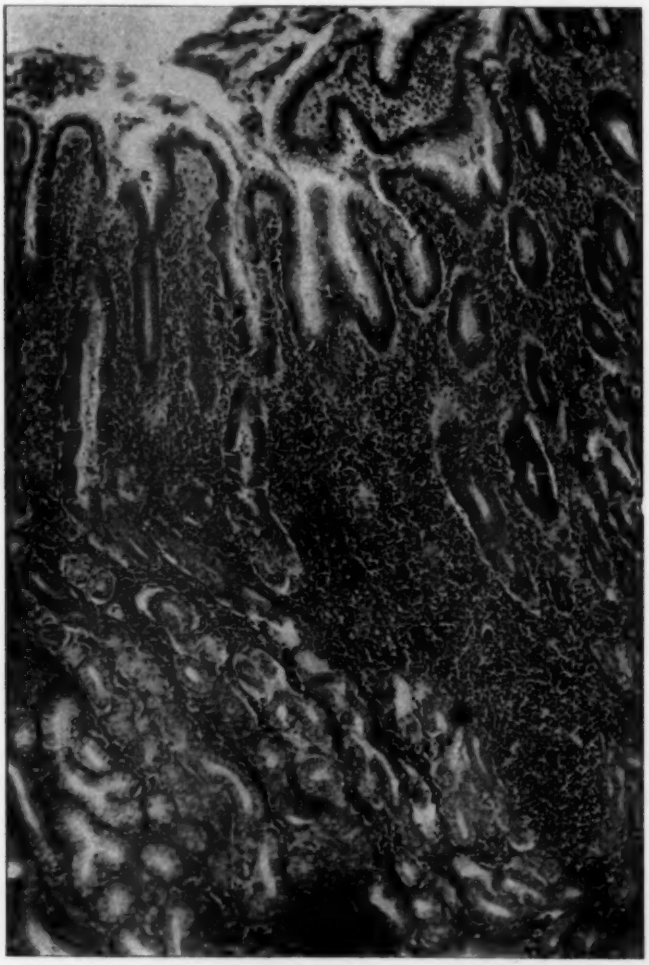


Fig. 4 (case 4).—Photomicrograph of a section made through the gastritic area shown in figure 2. Note the proliferation of the surface epithelium and the tremendous interstitial infiltration without alteration of the long gastric glands.

Dr. Palmer and his staff, we are unable to report on a greater number of cases in which operation was performed. In some cases we could follow this healing process through numerous gastroscopies, until complete epithelization took place.

Carcinomas.—CASE 5.—*History.*—The patient, aged 70, from the Cook County Hospital, was referred for gastroscopy by Dr. Harry Singer because of weakness and tarry stools.

Roentgen Examination.—The roentgenogram showed a small pancake-like infiltrative lesion of the prepyloric area with nichelike formation of the lesser curvature. It was uncertain whether the lesion was an ulcer or a carcinoma.

Gastroscopy (April 17, 1935).—Just beneath the angulus a small, jagged prominence was seen during inspiration, the color of which was darker than that of the angulus itself. The mucous membrane of the lesser curvature above the angulus was without marked changes, although we had the impression that it was stiff and infiltrated. In spite of the fact that the involved area was seen only for parts of a second, we were of the opinion that the condition must be a carcinoma of the lesser curvature of the antrum. The size of the tumor could not be estimated.

Operation (June 30).—Resection was done for carcinoma. Microscopic examination of the specimen showed ulcerative adenocarcinoma.

This case illustrates the difficulties of making a diagnosis even with gastroscopy. The lesion was situated in that part of the stomach which is the most difficult to see, i. e., the "blind" area of the lesser curvature of the antrum. A benign ulcer would never have been seen, although in some cases radial folds converging toward the angulus, causing the appearance of a sharp angle instead of the usual parabolic line, may indicate a hidden ulcer. In this case we were fortunate to see part of the lesion, even if for only a part of a second. This indicated with certainty that the lesion was malignant. Nevertheless, it must be admitted that a small carcinoma of this region may be overlooked, although we have never seen such a case.

CASE 6.—*History.*—A woman aged 38, seen on Oct. 12, 1934, complained of vomiting for the past two years from once to three times weekly. About three months before the present examination the vomiting increased in frequency, and two weeks before it occurred after almost every meal. The vomitus contained some undigested food and "brown foamy stuff" which tasted bitter. Recently this had been followed by burning pains around the umbilicus. The patient's appetite had been good, but for the past two weeks she had been afraid to take food because of the pain and vomiting which followed. There had been a loss of weight of 22 pounds (10 Kg.) in the last two months, unassociated with loss of strength. The patient was nervous and restless.

Physical Examination.—No abnormalities were found.

Laboratory Examination.—The red cell count was 5,040,000 and the white cell count 7,100. Gastric fluid obtained after an Ewald test meal showed: free acid, 9, and total acid, 106 (questionable). The stools gave a 4 plus reaction to the benzidine test for blood.

Roentgen Examination.—The roentgenogram showed a large penetrating crater on the lesser curvature of the stomach. The mucosal folds about this lesion and on the greater curvature were thickened and stiffened. The patient complained of marked tenderness over this area, and at times it seemed that an indefinite mass could be palpated. We were unable to differentiate definitely between ulcer

with associated regional gastritis or ulcer with associated carcinomatous infiltration. The impression was that there was a penetrating ulcer with associated regional gastritis. Carcinomatous infiltration could not be absolutely ruled out. The suggestion was made that the patient be referred for gastroscopy.

Gastroscopy.—A few centimeters above the angulus a large prominence of the lesser curvature was seen. An ulcer of irregular shape, with very irregular edges and surrounded by a definite wall, was found. There was no distinct edge, the ulcerated surface passing gradually into the mucous membrane of the wall, which was swollen and contained pigment spots.

On the basis of the examination it was decided that this was not the picture of a benign ulcer; a malignant lesion of the lesser curvature was suspected.

Operation (October 16).—A large carcinomatous mass was found involving the lesser curvature of the stomach extending almost to the cardia. Practically the entire stomach as well as a large section of the transverse mesocolon was resected. A posterior gastro-enterostomy was done between the first part of the jejunum and the small stump of the remaining portion of the stomach. Recovery was uneventful.

Pathologic Examination.—A crateriform ulcer, 4 by 3.5 by 1.5 cm., was seen. The base of the ulcer extended through much of the wall of the stomach and probably into the adherent omentum. Microscopic examination of a section through the margin of the large ulcerating lesion showed normal mucosa at the fundus which abruptly became necrotic, giving the typical appearance of the crater of an ulcer. Beneath the crater the wall of the stomach was composed of dense edematous fibrous tissue. The entire wall was infiltrated by carcinoma cells, varying greatly in size and appearance and showing areas of "colloidal" transformation with numerous signet ring cells. A section through the wall farthest from the lesion showed no carcinoma but a diffuse infiltration of the muscularis by polymorphonuclears.

In this case, in which the symptoms were of long duration, the roentgen examination could not establish a differential diagnosis between a benign and a malignant lesion. A benign lesion seemed to be more probable; however, the roentgenologist suggested the gastroscopic examination, which definitely proved the malignancy of the lesion. This close cooperation between the roentgenologist and the gastroscopist seems to us to show the proper attitude and the one which is to the best interests of the patient. Each method complements the other; they should most certainly not be regarded as methods of competition. Although in this case the development of a carcinoma from a benign ulcer cannot be proved, it is possible. It is quite certain that an earlier gastroscopic examination would have revealed the beginning carcinomatous formation. This case will again be mentioned in the section concerning operability of carcinomas and the value of early diagnosis in gastric carcinoma.

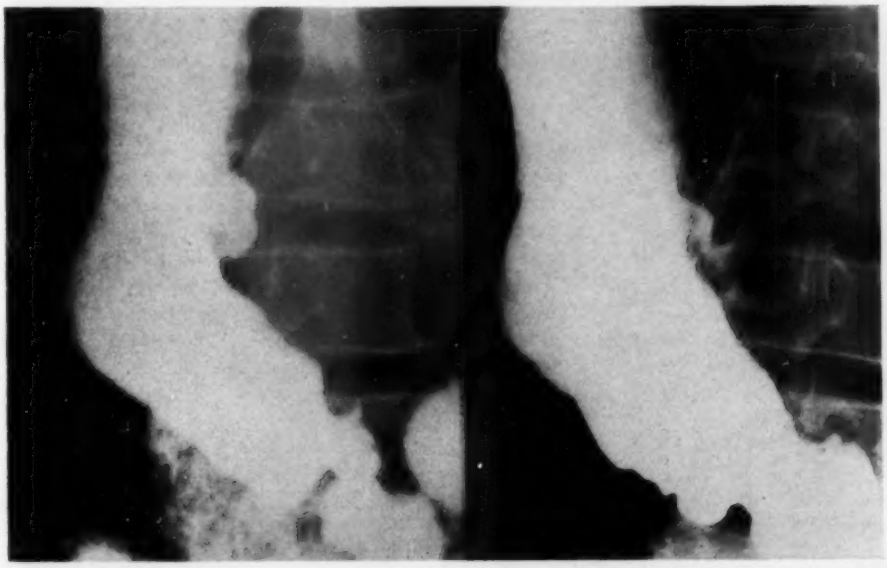
We consider the following the most important case presented in this paper.

CASE 7.—*History.*—A man aged 71, seen on June 4, 1935, three years before suffered from gnawing epigastric distress one or two hours after eating. A roent-

genogram demonstrated an ulcer, but the patient was not informed whether it was duodenal or gastric. He was placed on a diet, whereupon the distress disappeared. He had no further difficulties until about four months before the present examination, at which time he had daily distress which was relieved by the ingestion of powders or food. There was no loss of weight.

Physical Examination.—Bronchiectasis and obliterative pleuritis of the left side of the chest were found to be present. A roentgenogram of the heart showed the organ to be 29 per cent oversized. No mass could be palpated in the abdomen, and there was no tenderness.

Laboratory Examination.—The histamine test showed free acid (28) after thirty minutes. The stools gave a negative to a 4 plus reaction to the benzidine test for blood.



A B

Fig. 5 (case 7).—*A*, roentgenogram made on June 21, 1935. The large niche of the lesser curvature was believed to represent a benign ulcer. *B*, roentgenogram made nineteen days later. The markedly reduced size of the niche seemed to prove definitely the benign nature of the lesion. Compare with *C* and *D* in the color plate, *C* corresponding with figure 5 *A* and *D*, with figure 5 *B*.

Roentgen Examination.—We shall give the results of all examinations in connection with this case. It may be mentioned here that the fifth and sixth readings were proved to be incorrect. They were revised after comparison with the gross specimen.

June 17: A large penetrating ulcer of the cardiac end of the lesser curvature of the stomach was seen.

June 21: A benign penetrating ulcer of the cardiac end of the lesser curvature of the stomach was observed (fig. 5 *A*).

July 9: The crater was smaller (fig. 5 *B*).

July 19: The crater was becoming smaller.

August 9: The ulcer of the cardiac end was again becoming larger. A second lesion was present at the lower end of the lesser curvature of the stomach. One or both of the lesions were thought to be neoplastic.

August 26: The penetrating ulcer was still present. The second lesion was present at the lower end of the lesser curvature. The latter was probably carcinomatous.

October 31: There was no second lesion. The large crater was still present at the cardia of the lesser curvature.

First Gastroscopy (June 22).—A sharp picture of the lesser curvature was obtained. There was a large crater-like ulcer, the edge of which was rather sharp, but the whole cavity was filled with blood coagula. In this blood was seen a grayish ridgelike prominence. The mucous membrane surrounding the ulcer was partly thinned and partly swollen. On the basis of the gastroscopic findings a diagnosis of ulcer-like careinoma of the lesser curvature of the stomach was made. In spite of the high location, the carcinoma seemed to be so well limited that a radical operation was not completely out of the question. (See *C* in the color plate.)

Second Gastroscopy (July 8).—The pyloric region was instantly seen. About the entrance of the antrum the mucous membrane pattern was extremely irregular. On the anterior wall of the greater curvature there were nodes of varying size. On the lesser curvature, just above this point, an elevation was observed, on top of which was an ulcer. The edge of this ulcer lying toward the pylorus was not sharply defined, but all other edges were well demarcated. The floor was grayish white and was not bleeding. The slope of the elevation was extremely reddened and appeared to be edematous. The nodular changes at the entrance of the antrum were too large to permit a diagnosis of nodular gastritis. Carcinomatous infiltration was suspected. Furthermore, the ulcer of the lesser curvature did not have the appearance of the usual benign ulcer because of its situation on an elevated area and because one part of its edge was not sharply defined. For these reasons we believed that a carcinomatous lesion was present which was well limited toward the cardiac portion of the stomach but appeared to be progressive toward the pyloric part. Radical operation seemed to be possible at this stage. (See *D* in the color plate.)

Operation (November 4).—An ulcerating lesion was felt in the lesser curvature upward toward the cardia. About seven eighths of the stomach was resected The patient died on November 8 from bronchopneumonia. Permission for autopsy was not given.

Pathologic Examination.—Carcinoma was found on the lesser curvature. Microscopic examination showed colloidal carcinoma of the mucosa, well defined against the muscularis mucosae but invading the lymph vessels of the submucosa. The distant parts of the wall of the stomach showed dilatation of blood vessels and edema of the submucosa, which was three times thicker than usual (figs. 6 and 7).

This case demonstrates the superiority of gastroscopy over all other methods in arriving at this diagnosis. The history, and particularly the roentgenogram, suggested a benign lesion. Even after knowing of the presence of carcinoma, as proved microscopically, no one could find the least evidence of it either in or hidden by the mucosal folds, which the roentgenogram showed to be radiating from the ulcer. The pathologic examination offers the explanation: The carcinoma was almost noninvad-

ing. It did not even invade the muscularis mucosae. The gastroscopic picture, however, was definite, even after the first examination, at which the crater of the ulcer was seen filled with blood. It must be pointed out

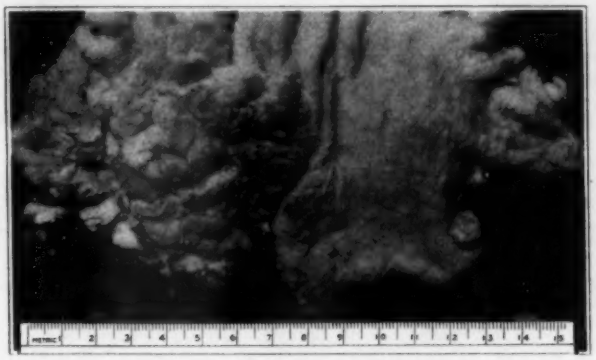


Fig. 6 (case 7).—Gross specimen of a carcinoma of the lesser curvature of the stomach after resection. Compare with *C* and *D* in the color plate. The resection was made four months after the second gastroscopy.

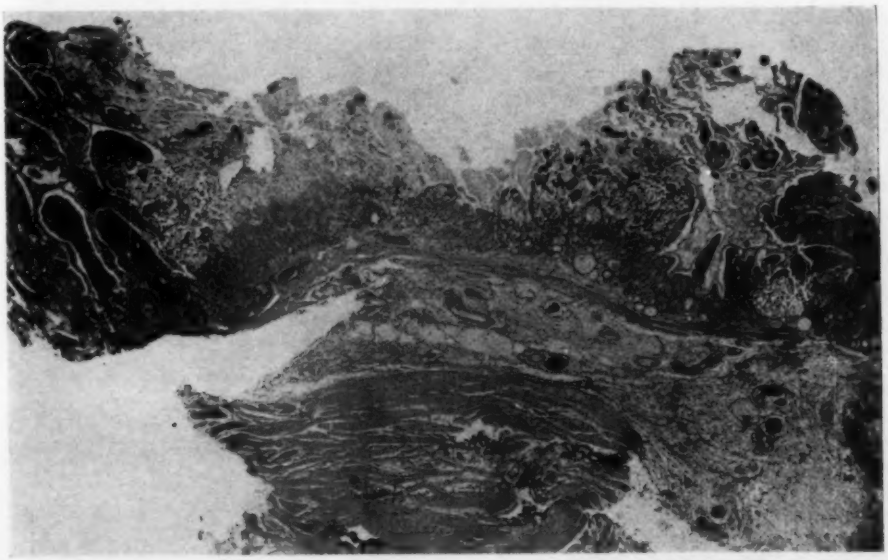


Fig. 7 (case 7).—Photomicrograph of a section made through the carcinoma shown in figure 6. Compare with *C* and *D* in the color plate. A colloid carcinoma of the mucosa is well defined against the muscularis mucosa but is invading the lymph vessels of the submucosa.

that much experience is necessary for such endoscopic accuracy; this is naturally also true of other diagnostic procedures. Here again it is possible, but it cannot be proved, that the carcinoma developed from a

preceding ulcer. The gastroscopic findings were so exact that even the enlargement of the mucosal blood vessels were seen (redness of the wall), and the edema of the gastric wall was described. The patient was not anxious to have an operation, and because he was a poor operative risk he was not urged too strongly. The fact that the niche in the roentgenogram became shallower during the observation while the carcinoma was progressive seemed to us to be important. Such cases have been described by Bloomfield.[3] It is obviously dangerous to assume that a niche is produced by a benign ulcer because it becomes shallower during roentgen observation of a few weeks; early gastroscopy is indispensable. We shall later discuss the fact that here a true "early" diagnosis of a gastric carcinoma was made by means of gastroscopy.

Conclusions.—Operation was performed on seven patients, in each of whom it was not quite certain clinically whether a benign or a malignant ulceration of the stomach was present. In four of these cases the ulcer was proved microscopically to be benign; in all of these cases the roentgen examination gave doubtful results, and in two carcinoma was thought to have been present. Gastroscopy established a correct diagnosis in every instance.

In three of the original cases the lesion was proved to be carcinomatous. The roentgen diagnosis was incorrect in one instance and uncertain in the remaining two. The gastroscopic diagnosis again was correct in each case. The case in which a malignant ulcer was diagnosed gastroscopically but in which the ulcer was thought to be benign, both by clinical impression and by the fact that the lesion became shallower on repeated roentgen examinations, will be discussed later. The splendid cooperation between the roentgenologist and the gastroscopist was appreciated as being of great value.

OPERABILITY OF CARCINOMA AS DETERMINED BY GASTROSCOPY

Inoperable Tumors.—The inoperability of a tumor is dependent not only on the local gastric findings but also on the general condition of the patient and on the presence or absence of metastases. Nevertheless, the inoperability may become evident instantly by gastroscopy. It may be shown that the tumor is not sharply defined and that the infiltration extends too high to allow even a radical operation. This can usually be demonstrated better by gastroscopy than by roentgen examination. We believe that no surgeon considers the exploratory operation satisfactory in cases in which the condition proves to be inoperable. Bier has said that exploratory laparotomy in cases of gastric cancer is murderous. We believe, however, that before the introduction of gastroscopy

3. Bloomfield, A. L.: Diagnosis of Early Cancerous Changes in Peptic Ulcer, J. A. M. A. **104**:1197-1201 (April 6) 1935.

exploration was warranted in many cases but that now it should seldom be necessary after careful examination by a well trained gastroscopist.

CASE 8.—*History.*—A man aged 64, seen on March 6, 1935, suffered from epigastric pain for ten weeks.

Physical Examination.—A hard movable mass was found in the left upper quadrant of the abdomen.

Laboratory Examination.—The gastric contents showed: free acid, 0, and total acid, 15.

Roentgen Examination (March 11).—A large gastric carcinoma, probably of the medullary type and probably located on the posterior wall, was thought to be present. The lesion extended high along the greater curvature, possibly so high as to be inoperable.

Gastroscopy (March 8).—The nodular angulus was overhanging the entrance of the antrum so that only a small portion of the antrum could be seen. The angulus and the lesser curvature above the angulus were stiff and infiltrated and showed numerous nodules. Color changes were present. The same findings were seen in the posterior wall, and, to a less extent, in the lesser curvature. There was no sharp limit of the mucous membrane, but we had the impression that from about 8 cm. beyond the cardia the mucous membrane was rather normal. On the basis of the gastroscopic findings a diagnosis of infiltrating cancer without sharp limits was made. In spite of the fact that the cancer was situated rather low in the stomach, its limits were not sharp, and because of this we believed that the chances of a successful operation were poor.

Operation.—A huge carcinoma was found involving the distal two thirds of the stomach. There were metastases along both the greater and the lesser curvature. The condition was considered inoperable. The patient died on March 18 of pulmonary embolism.

Autopsy.—Ulcerating carcinoma of the prepyloric area of the stomach was found arising in the posterior wall and permeating the wall of the stomach; it extended radially to the perigastric, pancreatic and periaortic lymph nodes from the level of the cardia to that of the renal vein, etc. Microscopic examination showed that the carcinoma had arisen in the gastric mucosa; it had spread through the lymphatics and permeated the wall of the stomach. The neoplasm consisted of atypical hyperchromatic epithelial cells which were growing in masses but with a definite tendency to tubule formation in places. The lymphatic spaces in the serosa of the stomach were dilated and filled with masses of carcinoma cells.

The carcinoma was considered inoperable because of both roentgen and gastroscopic observations; the latter showed infiltration and lack of sharp limitation of the lesion. Later the microscopic examination showed the diffuse infiltration of the wall of the stomach and the invasion of the lymphatic spaces of the serosa. Operation was attempted; the patient died from embolism.

CASE 9.—*History.*—A man aged 63, seen on Jan. 7, 1935, suffered from abdominal distress for eight months, with belching and regurgitation. There had been loss of appetite and weakness of about one year's duration.

Physical Examination.—An epigastric mass, unmovable and about 4 cm. in diameter, was felt.

Roentgen Examination (January 8).—The picture was that of polypoid carcinoma involving the central portion of the stomach. The extreme cardiac end and the pylorus were believed to be free from infiltration.

Gastroscopy (January 9).—A hard mass was felt a few centimeters below the cardia on the passage of the Ewald tube. The gastroscope could be introduced just beyond the cardia. In the lesser curvature a hemorrhagic infiltration was seen. The diagnosis was a large neoplasm of the upper part of the stomach, infiltrating the lesser curvature.

Operation (November 2).—A large thickened mass involving most of the greater curvature and most of the posterior wall and extending from the lower third to within about 2 cm. of the cardiac orifice was found. It was decided that an attempt at subtotal gastrectomy could not be successful since the stomach was adherent to the carcinoma, which had grown beyond the confines of the stomach. High voltage roentgen therapy improved the condition.

There was general agreement concerning the nature of the lesion. The carcinoma extended almost up to the cardia. The inoperability was best shown by gastroscopy. Such infiltrating lesions without sharp demarcation in the upper part of the stomach must always be considered as being unfavorable for operation, especially if the gastroscope cannot be completely introduced. It is questionable whether gastroscopy was not contraindicated in this case. Our diagnosis of infiltration of the lesser curvature of the stomach was shown to be incorrect. This case, with others, has influenced our new conception of gastroscopic orientation in the upper parts of the stomach.

Case 10.—*History.*—A patient aged 50, seen on April 24, 1935, suffered from weakness and a constant dull aching pain in the epigastrium for two months. He had lost 30 pounds (13.6 Kg.) in weight since Jan. 1, 1935.

Physical Examination.—No abnormalities were found.

Laboratory Examination.—The gastric fluid after the injection of histamine showed no free acid. The stools gave a 4 plus reaction to the benzidine test for blood. The blood showed 60 per cent hemoglobin and 4,000,000 red blood cells.

Roentgen Examination (April 26).—The roentgenogram showed a large carcinoma situated high on the lesser curvature of the stomach with a ragged crater at the center.

Gastroscopy (April 27).—At the cardia marked resistance was felt, but it was possible to bring the instrument into the stomach. Instantly a large tumor was seen on the anterior wall invading the lesser curvature. It extended from about the region of the angulus up to the cardia. The bleeding protrusion was not sharply limited and contained an irregular yellowish white ulceration. A diagnosis of a large carcinoma of the anterior wall and lesser curvature, not sharply limited, involving the cardia, was made. In our opinion the condition was inoperable but probably suitable for roentgen therapy.

Operation (April 30).—The stomach was examined and found to be involved by a carcinoma which extended along the lesser curvature, reaching up to the esophagus and infiltrating the gastrohepatic mesentery. Metastases were found in the liver. The involved portion of the stomach was fixed to the posterior parietes so that it could not be brought down and lifted out into the field. The patient was discharged on May 27 and died on July 3.

Roentgen examination and gastroscopy showed a carcinoma situated high up on the lesser curvature of the stomach. Gastroscopy gave evidence of its inoperability.

A 75 year old man had epigastric distress for one year, which was relieved by food. Physical examination gave essentially negative results except for a slight enlargement of the heart accompanied by an apical systolic murmur. The gastric fluid showed no free acid. The stools gave a 4 plus reaction to the benzidine test for blood. Roentgen examination revealed a penetrating ulcer of the lesser curvature of the stomach with infiltration of the lesser curvature (fig. 8). The roentgenologist felt that the extreme cardiac end of the stomach was not involved, but the examination was difficult because of the inability of the patient to relax. The gastroscopic findings follow: The pylorus and the antrum were about normal.

Fig. 8.—Roentgenogram in a case in which operation was not performed because gastroscopy (see *B* in the color plate) definitely proved the inoperability of the growth.

Just above the angulus a grayish white tumor appeared involving the lesser curvature, the anterior wall and the greater curvature. Protruding nodes were seen on the lesser curvature only. In other parts there were a whitish infiltration and rigidity of the entire mucous membrane. This picture was seen just below the cardia, although even the cardia was somewhat infiltrated; its mucous membrane was bleeding, and during the introduction of the instrument a slight resistance was felt at this point. A week later, as the chief of the gastro-enterologic department wanted to see the picture, gastroscopy was repeated. In this short time the changes had increased considerably, and the cardia was definitely involved; an ulceration was also seen. Dr. Palmer decided against a laparotomy after being shown these findings. In this case, again, gastroscopy showed much more detail than the roentgenogram. (See *B* in the color plate.)

Operable Tumors.—In this discussion we shall briefly refer to cases 5, 6 and 7 and present three others. In case 5 operability was shown after gastroscopic examination by the fact that the tumor was seen only on the angulus. Operability was deemed likely in case 6, but we could not be certain since the gastroscopic picture showed the edge of the ulcer passing into the normal mucous membrane. However, it could not be said that operation was out of the question. Practically the entire stomach and a large section of the transverse mesocolon were resected. Certainly the gastroscopic picture gave a more correct impression of the seriousness of the case than did the roentgen examination. In case 7, we stated, after gastroscopy, a half-year before the operation, that the tumor would be operable. If the patient had been convinced of the necessity of the operation earlier he might have survived, since the lesion was relatively benign and well confined. This case illustrates the obvious fact that an early diagnosis is of little value if the patient will not cooperate.

Case 11.—*History.*—A man aged 63, seen on Aug. 1, 1934, for the past twenty-five years had epigastric distress consisting of a localized burning pain occurring two or three hours after meal time and at about 2 a. m. This distress was always relieved by the ingestion of food. On two occasions his condition was diagnosed as a gastric ulcer. Since June 1933 his distress had changed somewhat. He complained of dull epigastric pain associated with fulness immediately after eating. His appetite had been poor, and he had lost 15 pounds (6.8 Kg.) in weight.

Physical Examination.—A questionable mass was felt in the epigastrium; otherwise the results of the examination were negative.

Laboratory Examination.—The gastric fluid obtained after an Ewald test meal showed: free acid, 29, and total acid, 75. The stools gave a 4 plus reaction to the benzidine test for blood.

Roentgen Examination.—The picture was that of carcinoma of the stomach with partial obstruction. The upper two thirds of the lesser curvature appeared pliable and did not appear to be infiltrated. The lower three fourths of the greater curvature also appeared to be pliable, with no infiltration. The patient would not give permission for operation. He was readmitted to the hospital on December 21.

Clinical Impression.—On the basis of the clinical picture the condition was thought to be a large carcinoma of the stomach, probably inoperable, but with no demonstrable metastases.

Gastroscopy (December 24).—The pylorus was instantly seen. It was a small, completely rigid hole, the posterior wall of which was covered by a prominent round tumor on which were seen two nodes. The border was well marked except for the posterior side, where no real edge could be seen. At this point there seemed to be a diffuse infiltration of the posterior wall where the mucous membrane was very thin, and a large blood vessel could be observed. The upper portions of the posterior wall, however, were quite normal, as were all the other portions of the mucous membrane except for being somewhat more pale than usual. The distal node of the tumor showed a pink and spotted mucous mem-

brane; it covered the pyloric channel, behind which an ulceration might possibly have been found. The proximal node was darker, and a large hemorrhage was seen in its mucous membrane. A diagnosis was made of carcinoma of the posterior wall of the antric channel infiltrating the lower posterior wall of the body of the stomach; nevertheless, it was thought that a radical operation could well be attempted because the tumor did not reach the upper parts of the stomach.

Operation (December 26).—The stomach was easily movable and could be brought up out of the abdomen. The carcinoma involved the entire circumference of the stomach close to the pylorus and had invaded the transverse mesocolon. Gastrectomy was done. The patient died of pneumonia on the second day after operation.

Pathologic Examination.—There was an ulcer crater measuring 4.8 by 3.3 by 0.8 cm., with sharp soft edges. It rested on fibrous tissue in which were found small nests of carcinoma cells. The wall adjacent to the ulcer was densely infiltrated with carcinoma cells arranged in long thin cords. A section through the wall of the stomach some distance from the carcinoma did not contain carcinoma cells but showed definite polymorphonuclear infiltration about the vessels of the anterior coat of muscle.

Autopsy.—No metastases were found.

The symptoms of long duration (twenty-five years) indicate that either an ulcer or chronic gastritis preceded the carcinoma. The clinical impression was that the condition was an inoperable carcinoma; the roentgenogram indicated that the tumor was movable; gastroscopy pointed out that a resection would be possible. The two last-mentioned impressions were found to be correct.

CASE 12.—*History.*—A 41 year old woman, seen on Nov. 13, 1934, suffered from pain in the lower half of the abdomen for two years, gnawing abdominal pain for the past eight months when the stomach was full and nausea and vomiting for the past three months.

Physical Examination.—A firm fixed mass was found in the abdomen which was painful on deep palpation. It was about 8 by 10 cm. in diameter and extended about 1½ inches (3.8 cm.) below the umbilicus.

Laboratory Examination.—The gastric fluid obtained after an Ewald test meal showed: free acid, from 28 to 60; total acid, from 40 to 70 and sarcinas. The stool gave a 3 plus reaction to the benzidine test for blood on one occasion.

First Gastroscopy (November 16).—On the greater curvature of the antrum just at the pylorus was seen a large ulcer with a dark brown floor and irregular edges, which were surrounded by an elevated reddened wall which seemed to be infiltrated but contained no nodules. Peristaltic waves were seen on the side of the lesser curvature passing toward the pylorus. The upper parts of the mucous membrane were normal except for one small area high on the posterior wall, where some edema was seen, in the middle of which was found a small but definite dimple-like ulcer with elevated edges and a brownish floor. On the basis of the gastroscopic findings a diagnosis was made of cancer of the pylorus and the greater curvature, which was sharply limited (which probably could be radically resected), and circumscript hypertrophic gastritis of the upper posterior wall with a small ulceration, which was probably metastasis to the mucous membrane.

Roentgen Examination (November 16).—The roentgen picture was that of annular carcinoma of the antrum of the stomach. An inflammatory lesion with massive adhesions about the stomach could not be ruled out, but the possibility of such a diagnosis was doubtful.

Operation (November 17).—Carcinoma was found in a movable stomach. The pyloric region was involved, the glands along the pancreas were enlarged and one small metastasis was found at the site of the gallbladder. Since resection was easy, it was done in preference to gastro-enterostomy because of the greater palliative relief. About half of the stomach was resected.

Pathologic Examination.—A large ulcerous crater was seen lying in carcinomatous tissue with irregular, firm borders and measuring 4 by 1.5 cm. Microscopic examination showed normal mucosa along one border, which abruptly ran into the superficially necrotic ulcer bed. Beneath the normal mucosa and the ulceration, the normal structures were replaced by large irregular areas of typical small groups of carcinoma cells containing colloid and being of the signet ring type. High voltage roentgen therapy was followed by recovery.

Second Gastroscopy (December 20).—Most severe inflammation of the whole mucous membrane was seen, certainly due to the roentgen treatment. Metastases could not be seen.

Roentgen examination revealed the carcinoma in this case; there were some doubts concerning the lesion, however. Gastroscopy showed the carcinoma with the greatest certainty; it further indicated the advisability of resection because of the sharp limits of the tumor. The presence of metastases as a probability was also mentioned. All these observations were confirmed by operation.

CASE 13.—*History.*—A 50 year old man, seen on Aug. 2, 1935, had suffered from gaseous distention and loss of weight for the past eight months.

Physical Examination.—Resistance was encountered in the epigastrium, and a systolic murmur was heard.

Laboratory Examination.—The stools were repeatedly normal.

Gastroscopy (August 5).—Some food remnants were present in the contents of the stomach obtained during fasting. There was an irregular, central, elevated, granular wall which was slightly more red than usual, even having a brownish tinge, and in the middle of which was seen a dark crater. The surrounding mucous membrane showed no significant changes; the upper parts of the stomach were normal. A diagnosis was made of circular carcinoma of the pylorus, well limited. It was thought likely that a radical operation could be performed successfully. (The growth was so typical of an operable carcinoma that we present here a reproduction of the gastroscopic picture [*E* in the color plate], together with a photograph of the gross and microscopic specimens.)

Roentgen Examination (August 5).—The roentgen picture was that of neoplastic involvement of the prepyloric portion of the stomach by a constricting and apparently polypoid type of tumor.

Operation (August 5).—The pyloric region was found to be involved by a hard tumor mass the size of a walnut. Several hard lymph nodes were present at the lesser and greater curvatures. Subtotal gastrectomy was done (because of the lymph nodes). The patient died from postoperative hemorrhage.

Pathologic Examination.—The gross specimen presented almost the exact picture that was seen by gastroscopy. Microscopic examination showed the cells of

the mucosa to be atypical, being arranged in cords, with alveoli deeply infiltrating the wall of the stomach. There was no tumor in the cardiac end. The lymph nodes showed evidence of carcinoma (figs. 9 and 10).

Subtotal gastrectomy was done because of the presence of the involved lypmh nodes. The neoplasm was as sharply limited as gastroscopy had shown it to be. It is probable that there was no carcinomatous invasion of the lymph nodes a few months earlier, and if the patient had been seen at that time his life might have been saved. This illustrates the great importance of early gastroscopic examination, since the malignant nature of even very early lesions can thus be established.

Conclusions.—Nine cases of gastric carcinoma were observed in which the question of operability arose. In three of these cases the condition was found at operation to be inoperable. In two of them roentgen examination and gastroscopy were in agreement concerning the impossibility of a radical operation. The condition in the remaining case was also found to be inoperable by gastroscopy; the roengenographic protocol gave no opinion concerning this point.

A radical operation was performed in the remaining six cases; in three the condition was found to be definitely operable after gastroscopic examination. Not only was this true, but the findings at operation were almost identical with those described in the gastroscopic protocols. The final three cases presented complete agreement in only one instance between the roentgen examination and the gastroscopic examination; here both methods demonstrated the operability of the lesion. The fifth lesion was shown by the roentgenogram to be operable, whereas the gastroscopist stated that though the original growth was definitely operable he believed metastases to have taken place. This was found to be true. In the remaining case differentiation between a benign and a malignant ulcer could not be made roentgenographically. Gastroscopy answered this question, and, moreover, the results of operation were considered doubtful. This was borne out by the finding of metastatic lymph nodes, which necessitated subtotal gastrectomy.

VALUE OF GASTROSCOPY IN THE "EARLY" DIAGNOSIS OF GASTRIC CARCINOMA [4]

We shall consider an "early" diagnosis of carcinoma of the stomach as being one which makes it possible to bring about a cure of from three to five years' duration. Although operation must be considered the only method of cure, high voltage roentgen therapy by the Coutard method may be tried in cases in which the lesion is inoperable. Curiously, general practitioners, and even gastro-enterologists, are found who do

4. Refer to cases 7 and 29. A more extensive discussion of this question will be found in the senior author's book on "Gastroscopy" (Chicago, University of Chicago Press, 1937), which contains ninety-six colored pictures of gastroscopic findings.

Fig. 9 (case 13).—Resected gross specimen of the carcinoma. Compare with *E* in color plate. The growth is a typical sharply limited operable carcinoma.

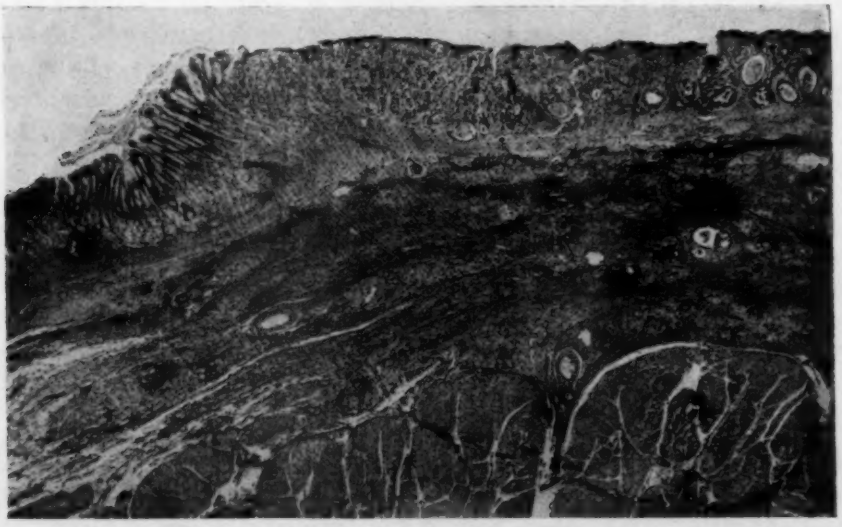


Fig. 10 (case 13).—Photomicrograph of a section made through the carcinoma shown in figure 9. Note the sharp demarcation between the tumor (at the right) and the normal gastric glands (at the left).

not believe that good results are often obtained from gastric resection in cases of carcinoma of the stomach. One of us [5] has followed three patients for over ten years after this operation. Only one of them is known to have died, death resulting from a recurrence eleven years later. Numerous cases were observed in Munich in which the operation resulted in complete cure for from three to five years. Recent German statistics concerning the success of gastric resection in cases of carcinoma give a surprisingly high percentage of cures; it is unfortunate that the records are not available to us. This success was obtained because the cases were well chosen.

We believe that exploratory laparotomy is responsible, to a great measure, for the attitude common among the laymen and many medical men that in general operation for cancer of the stomach is hopeless. This attitude is not so difficult to understand. These persons are cognizant of the fact that even though operation has been attempted, in cases of advanced gastric carcinoma the ultimate prognosis is seldom better than before, and they believe this result obtains after surgical intervention in all cases of carcinoma of the stomach. It is our contention that if exploratory laparotomy could be done away with, except in those relatively few cases in which gastroscopy does not settle the question definitely, the mortality would be decreased to the point where patients would be willing to undergo operation. They would learn that early cancer can be cured by this procedure, whereas it is not even attempted on late cancer. In short, they may be brought to realize that the prognosis of early cancer is a different thing from the prognosis of late cancer. This would result, in our opinion, not only in more operations in cases of gastric carcinoma, but particularly in a higher percentage of operations in cases of the early stage in which definite cure is most likely to be brought about.

Boas said that by the time symptoms are present it is almost invariably too late to bring about a cure in cases of gastric carcinoma. This statement, frequently repeated, is not correct. If we control the few cases cited here we find histories of distress for periods of ten weeks, five months, eight months (two cases), two years (two cases) and twenty-five years (one case). In this last case it was probable that a benign ulcer was first present and that the carcinoma developed within the past six months. It is true that there are cases in which the lesion is inoperable fourteen days after the first symptom is noticed, but they are not frequent. Often early symptoms are present, or a history of

5. Schindler, R.: Die Bedeutung der Gastroskopie für die Heilung des Magenkrebses, Monatschr. f. Krebsbekämpf. **1**:203-208 (May) 1933.

symptoms of long standing (ulcer, atrophic gastritis, benign tumor) precedes the beginning of the carcinoma. Also, we must admit that there are types of carcinoma for which the prognosis is bad even with early diagnosis and operation. This is illustrated by the type which diffusely infiltrates the body of the stomach. The sharply limited tumors, even though they are of upper parts of the stomach, give good surgical results if the operation is done before the lymph nodes have been involved.

In order to improve the results of operation for gastric carcinoma and in order to dissipate the unjustified distrust which patients and even physicians feel regarding this procedure, we offer the following three suggestions:

(*a*) Patients over 35 years of age who have anorexia and loss of weight should be examined roentgenographically and gastroscopically, since it is at this stage of a carcinoma that the best surgical results are to be had. Of course, patients of this age having frank gastric symptoms which are not completely relieved after fourteen days of medical treatment should also be given a thorough examination, including the taking of roentgenograms of the gastro-intestinal tract and gastroscopy.

(*b*) All patients suffering from a gastric carcinoma which is not diffusely infiltrating but is sharply limited should be advised to undergo operation regardless of the location of the neoplasm, unless it is at the extreme cardiac end. On the other hand, a diffusely infiltrating tumor should be operated on only if it is small and if it is strictly confined to the pyloric region.

(*c*) Exploratory laparotomy should be done only in the relatively few cases in which gastroscopy cannot definitely decide the operability of the lesion.

Considering the first suggestion, we might ask what constitutes a "thorough" examination of the stomach. Certainly analyses of the gastric fluid and stools are not sufficient; in the small well limited tumor free acid is often found but occult blood is seldom seen. There are, then, only two methods of great value in this respect: roentgenography and gastroscopy. It is worth while to consider which gives the more valuable information concerning gastric carcinoma. If case 7 is analyzed it is seen that at least in that instance gastroscopy was much superior. This was a true case of "early" diagnosis; the carcinoma was recognized when it was extremely small. The roentgenogram demonstrated the niche but failed to demonstrate its malignant character. (This error was frequently seen in the material from Munich.) Roentgenography is able to indicate the malignant character of an ulcerative lesion if some infiltration has occurred around the lesion, and even then it is difficult to differentiate between inflammatory reaction and neoplastic infiltration. Gastroscopy, however, demonstrates the malignant

nature definitely to the trained gastroscopist. Moreover, in polypoid types of tumors gastroscopy can show the malignant development at its inception, while this is often impossible by roentgenography. One of us had the occasion to perform gastroscopy on a patient in Bergmann's Clinic (*charité*) in Berlin whose roentgenograms were suggestive of a benign tumor of the greater curvature; gastroscopy revealed a malignant, decaying, sharply limited carcinoma.

Roentgenographic examination, on the other hand, should theoretically be superior to gastroscopy in some cases since there are certain portions of the stomach "blind spots" which cannot be seen by the latter method. These regions of the stomach which can sometimes not be seen are the lesser curvature of the antrum and a small strip of the posterior wall on which the instrument lies. A small beginning carcinoma should be seen there in some instances when the gastroscope fails to demonstrate the lesion. (Although this is theoretically true, it is interesting to note that Schindler has not seen an instance of this although he has had occasion to observe twenty-four hundred gastroscopies and about six thousand roentgenograms.) Small diffuse carcinomas are difficult to demonstrate by either method; these growths, however, are usually so malignant that even early diagnosis is not of great value in prolonging life.

Conclusions.—The physician and the patient should be convinced of the value of operation for gastric carcinoma. To accomplish this, we suggest that exploratory laparotomy be done only in those few cases in which gastroscopic examination does not definitely settle the question of operability.

Surgical intervention can be done at an early stage if an early diagnosis is made; this, we believe, can be brought about by having every patient over 35 years of age examined both by gastroscopy and by roentgenography if he has been suffering from anorexia, abdominal distress and suggestive loss of weight. We further believe that such patients should be followed and examined gastroscopically and roentgenographically at least twice a year, since there is unanimity of opinion (Konjetzny,[6] Hurst,[7] Henning,[8] Moutier and others) that atrophic gastritis and benign tumor may develop into carcinoma.

No gastric ulcer should be treated without a preceding gastroscopic examination, and this examination should be repeated in those cases in

6. Konjetzny, G. E.: Ueber die Beziehungen der chronischen Gastritis mit ihren Folgeerscheinungen und des chronischen Magenulcus zur Entwicklung des Magenkrebses, Beitr. z. klin. Chir. **85**:455-519, 1913.

7. Hurst, A. F.: Alvarez Lecture on Unity of Gastric Disorders, Brit. M. J. **2**:89-94 (July 15) 1933.

8. Henning, N.: Die Entzündung des Magens, Leipzig, Johann Ambrosius Barth, 1934.

which there is a change in the type of distress, even if the niche as seen in the roentgenogram has become shallower, since this observation does not exclude the possibility of malignancy (see case 7, and compare with Bloomfield's case [8]).

By following this program we can hope to improve the results of operation on gastric neoplasms. This will result in increased confidence of physicians and patients in surgical intervention and will make it possible to bring not only a larger number of patients to the operating table but particularly a higher percentage of patients with early lesions. We may hope in this way greatly to improve the prognosis of the most frequent carcinoma seen in man.

GASTROSCOPY OF THE POSTOPERATIVE STOMACH

There are four principal morphologic changes which may cause trouble (pains, hemorrhages and belching) after gastro-enterostomy or gastric resection. They may be listed as follows:

1. Recurrent or gastrojejunal ulcer. This condition was long thought to be the only complication in the postoperative stomach.

2. Chronic gastritis. The frequency of this postoperative condition was first noted by one of us (R. S.) in 1922; it was then confirmed by all recognized gastroscopists (Korbsch,[9] Hohlweg,[10] Gutzeit, Hübner,[11] Hertel and Kallius,[12] Henning and others) and later by roentgenologists and finally by outstanding surgeons.[13]

3. Erosions and irritation caused by silk sutures remaining from operation, which have cut through the mucous membrane. The importance of this condition was also first noted by one of us [14] and was later confirmed by Henning and Moutier.

4. Carcinoma which develops at the stoma. This condition is rare but has been described; we have observed only two cases gastroscopically.

In our gastroscopic material we present thirty cases in which postoperative examination was made. In seven of these reoperation was done after gastroscopy, thus giving us a biopsic control. We shall first

9. Korbsch, R.: Zur Kenntnis der chronischen Gastritis, Med. Klin. **21**:959-962 (June 26) 1925.

10. Hohlweg, H.: Beobachtungen und Betrachtungen auf Grund von 100 Gastroskopien, München. med. Wchnschr. **71**:506-508 (April 18) 1924.

11. Hübner, A.: Gastroskopie, Ergebn. d. Chir. u. Orthop. **20**:266, 1927.

12. Hertel, E., and Kallius, H. U.: Beiträge zur Gastroscopie des operierten Magens, Arch. f. klin. Chir. **151**:578-594, 1928.

13. Clairmont, P.: Die Gastritis nach Magenoperationen, München. med. Wchnschr. **73**:1906 (Nov. 5) 1926.

14. Schindler, R.: Die diagnostische Bedeutung der Gastroskopie, München. med. Wchnschr. **69**:535-537 (April 14) 1922.

present this group. The remaining twenty-one cases shall be presented briefly, since we have no objective evidence of the accuracy of the gastroscopic findings.

POSTOPERATIVE STOMACH WITH BIOPSIC CONTROL

CASE 14.—*History.*—A 37 year old man, seen on Nov. 16, 1934, in 1924 had had an operation for gastric ulcer. In 1933 he began to complain of pain in the lower abdominal quadrants. The second operation, done on November 24, revealed a jejunal ulcer. Resection of the respective parts of the stomach and the jejunum was done, and a lateral anastomosis was made between the large opening of the stomach and a portion of the jejunum about 5 inches (12.7 cm.) below the old opening. After this, the patient again complained of abdominal pain.

Physical Examination.—The liver was enlarged, but there were no other significant findings.

Laboratory Examination.—Gastric analysis after the injection of histamine showed free acid up to 90. The stools gave a negative to a 3 plus reaction to the benzidine test for blood.

Roentgen Examination (Jan. 18, 1935).—The gastro-enterostomy stoma functioned well. At times a fleck was seen, about 5 or 6 mm. in diameter, near or in the stoma, which seemed to be definite, constant and circumscribed. It must be admitted, however, that its exact nature was not definite, i. e., it was not known whether it was barium in the crater of an ulcer or a bead of barium in the gastro-enterostomy stoma. There was little tenderness over it.

Gastroscopy (January 22).—The antrum was easily seen; cross-folds were running over its greater curvature. The gastro-enterostomy stoma was found lying in the posterior wall. It was a rather round hole from which some grayish fluid passed into the stomach during the patient's respirations. No peristalsis was seen, either of the opening or of the intestine. On the edge of the stoma toward the pylorus there was a deep crater-like ulcer the diameter of which was larger than that of the artificial opening. Its edges were sharp, and its floor was yellowish but somewhat spotted. That part of the ulcer which lay on the side of the anterior wall of the stomach was undermined and made a shadow on the floor of the ulcer. On the opposite side a black silk thread was seen which was hanging from the posterior wall of the gastro-enterostomy stoma. The folds on the cardiac side of the stoma were reddened and somewhat swollen; beyond this the mucous membrane as well as all the other parts of the stomach were normal. The diagnosis on the basis of the gastroscopic findings was a large marginal ulcer at the pyloric side of the posterior gastro-enterostomy stoma and slight gastritis of a small area surrounding the stoma. The silk thread near the stoma had cut through the mucous membrane. (See *F* in the color plate.)

Operation (January 28).—A large penetrating ulcer was found in the jejunum about 1 cm. from the suture line. The junction of the jejunum and the stomach was completely excised. A new side to side anastomosis of the jejunum was made.

Pathologic Examination.—The crater of the large ulcer was seen; also a long black silk thread was found extending into the gastric cavity. Microscopic examination showed diffuse leukocytic infiltration and foci of large foreign body giant cells and fat-filled macrophages.

The patient was examined roentgenographically and gastroscopically after his second operation for gastrojejunal ulcer. At fluoroscopy the

lesion was thought to have been seen but with no degree of certainty, whereas it was easily and clearly shown by gastroscopy. In regard to the fluoroscopist's statement that the stoma functioned well, it may be best to add here that a gastro-enterostomy stoma is said by the fluoroscopist to function well if the opening is patent. The gastroscopist speaks of normal function if the opening is patent and if rhythmic pylorus-like contractions are observed at the stoma. The fact that the opening allows the passage of food is not in itself sufficient. At the operation the ulcer was thought to be in the jejunum, 1 cm. from the stoma, whereas it was called a marginal ulcer at gastroscopy. In this case we are more inclined to believe the gastroscopic picture since these ulcers are always embedded in adhesions, and thus their exact location is often difficult to determine at operation. Moreover, it is difficult to believe that the ulcer could have been seen so easily and to such a full extent had it not been situated on the marginal line. The finding of the silk sutures was important and will be discussed later.

Case 15.—*History.*—A 31 year old man, seen on Aug. 30, 1934, had in 1924, because of a diagnosis of gastric ulcer, been on a Sippy diet, with marked improvement in his condition. The following year he had several hemorrhages, and in 1926 a gastro-enterostomy was done. In 1933 an emergency operation was performed because of perforation of the old ulcer. Since Aug. 26, 1934, he had had severe abdominal distress, tarry stools being noted occasionally.

Physical Examination.—Tenderness was noted in the epigastrium.

Roentgen Examination.—The roentgen picture offered suggestive but not conclusive evidence of gastro-jejunal ulcer. Seven gastroscopic examinations were done on this patient; one of them preceded a new operation, while the others followed.

Gastroscopy (September 6).—The antrum was well seen. The gastro-enterostomy stoma was hidden behind a fold of the posterior wall. Radial folds were seen converging toward this point. At a distance of about 1 cm. from the edge of the stoma a small flat grayish yellow ulcer was seen. The surrounding area was not inflamed. On the basis of the gastroscopic findings a diagnosis was made of flat erosion of the posterior wall near the gastro-enterostomy stoma but no associated gastritis.

Operation (September 30).—Extensive adhesions were found. The stomach had previously been divided at the pylorus and anastomosed into the jejunum by side to end anastomosis. No ulcer was found. Partial gastrectomy was done.

Pathologic Examination.—No visible ulceration was present. The mucosa was edematous, and there were small submucous hemorrhages just proximal to the connective tissue scar.

The patient was seen again on December 28 complaining of "sour belching" and pain between the shoulders. Roentgenography did not show any pathologic process. There were intervals when the patient felt quite well, but the distress would frequently recur. The histamine test at that time showed free acid up to 30. We shall briefly list the findings at gastroscopies performed on the following dates:

November 28: It was possible to see the interior of the jejunum. The folds running toward the edge of the gastro-enterostomy stoma were purplish red and

definitely swollen; two shallow erosions were seen. All other parts of the mucous membrane were also swollen; in the parts near the cardia hemorrhages were seen.

December 12: The erosions previously described had disappeared, but the mucosa surrounding the artificial opening was still extremely swollen.

January 29: The stomach was so sensitive that it retained with difficulty the air introduced during the examination. The area surrounding the stoma was extremely red and covered with small nodules.

May 28: The color of the mucosa of the jejunum did not show its usual uniformity; there were many grayish spots in the red mucosa. Small nodules were seen in the suture line.

July 1: The inflammation of the jejunum and stomach showed great improvement.

October 23: The artificial opening seemed to be narrower than at the last examination. The borders were definitely reddened. Thick layers of gray mucus were seen firmly adherent to the surface of the entire body of the stomach. Just below the cardia some thickened nodules were seen.

This patient was observed through a series of seven gastroscopic examinations. Before his admission to the hospital there had been several hemorrhages. Two operations had been done, the second being for perforation of an old ulcer. At the first gastroscopy the anastomosis between the end of the stomach and the jejunum was mistaken for the pylorus. After operation the stomach is often greatly distorted, making orientation difficult; such mistakes are not infrequent. A small flat yellow erosion was seen which we believe does not correspond with the roentgen picture since small erosions cannot be seen roentgenographically. Resection was justified because these erosions frequently are the cause of extensive hemorrhages. These extremely small lesions could not be seen in the gross specimen because the lack of circulating blood made the color a uniform grayish red. Gastroscopic visualization of the erosion and the definite history of hemorrhages precluded any possibility of doubt as to the existence of the lesion. The resection was followed by the development of severe gastritis, which was observed over a period of eleven months. It improved somewhat but never completely healed.

CASE 16.—*History.*—A patient of Drs. J. Meyer and Necheles, at the Michael Reese Hospital, was seen on May 27, 1935. Gastro-enterostomy was done four years before for ulcer. The patient's complaint at the time of the present examination was distress typical of that produced by ulcer.

Gastroscopy.—The pyloric region was seen. Much secretion containing air bubbles was seen passing into the antrum from the duodenum. The greater curvature of the antrum presented several small yellow and orange-red spots. Above the angulus on the lesser curvature larger red spots were seen. The gastroenterostomy stoma was found on the posterior wall; the margin was well seen and did not show the presence of an ulcer. A true jejunal ulcer, however, could not be ruled out. The diagnosis on the basis of the gastroscopic picture was severe superficial gastritis, especially of the greater curvature of the antrum and of the upper parts of the lesser curvature.

Operation (Dr. Strauss).—No ulcer was felt. A resection was carried out.

Pathologic Examination.—The gross examination (Dr. Saphir) revealed a gastro-enteric orifice measuring .4 cm. in diameter; the serosa in this region contained fine grayish white streaks. The mucosa of the stomach was smooth, pinkish gray and glistening. Minute petechial hemorrhages were present in various areas, and the mucosa of the jejunum was dark red and hemorrhagic. Microscopic examination (Dr. Schindler) revealed in some portions of the specimen thickened and dense connective tissue below the surface epithelium; in other areas an extensive cellular infiltration was seen between the upper layers of the mucosa. In some spots there was lymphocytic and plasma cell infiltration. The capillaries of only the most superficial part of the mucosa were found to be much enlarged. Some cells of the glands showed cloudy swelling and vacuolation.

This patient had had gastro-enterostomy performed four years before and was at the time of examination complaining of symptoms suggestive of ulcer; there was also a psychoneurotic element present. Gastroscopic examination demonstrated severe chronic gastritis but no ulcer. This finding is frequent in the postoperative stomach. The red spots which were seen were well explained by the greatly enlarged blood capillaries of the superficial parts of the mucosa.

CASE 17.—*History.*—A 52 year old man, seen on July 7, 1934, had been placed on medical management in 1930 because of a diagnosis of duodenal ulcer with moderate stenosis. In January 1931 gastro-enterostomy was done. In February 1933 the patient had a hemorrhage from a gastrojejunal ulcer, which again occurred on July 10, 1934. He was placed on medical management but still complained of a gnawing sensation in the epigastrium, and on December 10 hemorrhage occurred again.

Physical Examination.—The patient appeared to be pale. A small area of tenderness was found in the midepigastric region.

Laboratory Examination.—The blood showed 55 per cent hemoglobin and 3,425,000 red blood cells. The stools gave a 1 to a 4 plus reaction to the benzidine test for blood.

First Gastroscopy (December 21).—The gastro-enterostomy stoma was not found with the retrograde instrument. In the normal mucous membrane of the cordlike musculus sphincter antri two small hemorrhages were seen. There were many nodules on the lower parts of the posterior wall, and the mucous membrane in this region was reddish gray and contained numerous red spots. The opening of the jejunum was not seen. The diagnosis on the basis of the gastroscopic picture was hemorrhages in a normal mucous membrane and severe chronic gastritis of the lower parts of the posterior wall.

Second Gastroscopy (December 27).—This time the usual straight tipped instrument was used, and the artificial opening was instantly found. It was possible to see the greater part of its edge and to look into the jejunum. The inflammation, seen on previous examination, was definitely improved. There was a polyp-like node overhanging the upper edge of the artificial opening, the mucous membrane of which was somewhat swollen and extremely reddened.

Roentgen Examination (December 28).—No jejunal ulcer was seen.

Second Operation (Jan. 2, 1935).—No ulcer was found. The line of anastomosis between the stomach and the jejunum was cut away. The artificial stoma was closed, and the ends of the divided jejunum were anastomosed.

Pathologic Examination.—The line of anastomosis showed no inflammation in the msucular wall, nor did the sections demonstrate an ulcer. Microscopic examination (Dr. Schindler) showed extensive interstitial infiltration of the mucosa with plasma cells, round cells and some eosinophils. The cells of the surface epithelium showed vacuolation and cloudy swelling. In certain areas the capillaries in the superficial layers of the mucosa were much enlarged. Occasional rupture of the vessels was seen with resultant hemorrhages. Infiltration of the submucosa was noted in some areas (fig. 11).

Third Operation (January 12).—Another operation became necessary because of the formation of a dense band of fibrous tissue, causing almost complete obstruction of the first part of the jejunum. Recovery was uneventful.

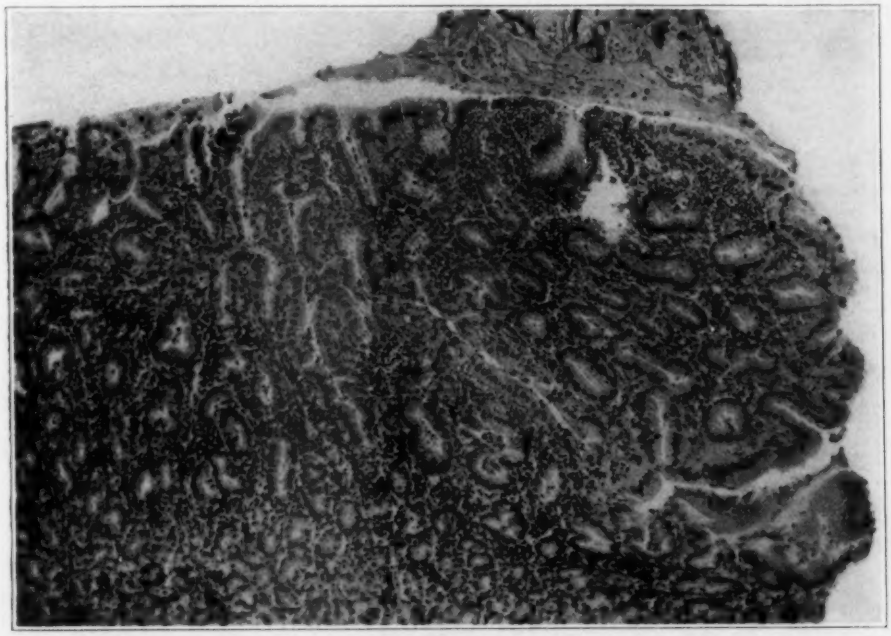


Fig. 11 (case 17).—Photomicrograph of a section made through the gastric mucosa after reoperation. Interstitial infiltration and exudation are well seen. The enlarged capillaries, strikingly seen in the microscopic specimen, do not show well in the reproduction.

Nine months later the patient complained of nausea and vomiting. He was placed on the Winkelstein treatment and was reported on November 8 to have been doing well.

Roentgen Examination.—The antrum showed a deformity which was probably the result of the old operation.

Third Gastroscopy.—An artificial opening was seen toward which folds of mucous membrane were converging. Above the opening one pigment spot and several small red spots were seen. The diagnosis on the basis of the gastroscopic picture was a postoperative stomach with a functioning stoma. The general con-

dition showed surprising improvement after the second operation. Small hemorrhages and one pigment spot were seen in normal mucous membrane.

The patient originally had a duodenal ulcer, for which a gastro-enterostomy was done. Several hemorrhages occurred during the following four years. No ulceration could be found by roentgen examination or gastroscopy or at operation. Severe gastritis was then demonstrated by gastroscopy. After this the gastro-enterostomy stoma was closed; at the operation the ulcer was found to be healed. At the third gastroscopy the severe gastritis found previously had almost entirely healed. The mucous membrane, however, showed changes which were consistent with an ulcer of the stomach. The pigment spot should be considered as being a marked preulcerative or postulcerative condition. This case demonstrates a fact that we have previously observed; that even extensive hemorrhages occurring in a postoperative stomach may be caused by a severe gastritis which is frequently mistaken for an ulcer. Moreover, this case demonstrates that postoperative hemorrhagic gastritis may heal if the gastro-enterostomy stoma is closed.

CASE 18.—*History.*—A 53 year old woman had had gastro-enterostomy done nineteen years previously for gastric ulcer. Her present complaint was localized epigastric pains.

Physical Examination.—There was moderate tenderness in the left upper quadrant 2.5 cm. from the umbilicus, but no other significant findings.

Laboratory Examination.—The stools gave a negative to a 3 plus reaction to the benzidine test for blood.

Gastroscopy (July 22).—The stomach was well seen. The angulus was so situated as to allow us to see the entire lesser curvature of the antrum. Just above the musculus sphincter antri on the anterior wall several small nodules were seen. At a higher level on the posterior wall the gastro-enterostomy stoma was seen. It could not be well observed since it was filled with a secretion of bloody mucus and air bubbles which was being expelled into the stomach from the jejunum. The presence of a gastrojejunal ulcer could not definitely be determined. The upper parts of the anterior wall were markedly swollen and contained small hemorrhages and nodules. The diagnosis on the basis of the gastroscopic picture was nodular hemorrhagic gastritis in a postoperative stomach. There was a bloody secretion from the gastro-enterostomy stoma.

Roentgen Examination (July 6).—Two of the views suggested the presence of a small fleck on the jejunal side of the gastro-enterostomy stoma. The rugous folds of the jejunum were thickened and distorted. Flecks at the gastrojejunal opening were extremely hard to evaluate because of operative pockets, but in view of the clinical findings we saw no reason to doubt that the fleck seen at fluoroscopy and on the films was anything other than the crater of an ulcer. The picture suggested gastrojejunal ulcer with jejunitis.

Operation (August 7).—An indurated area of a diameter of 1 cm. could be felt on the distal loop of the jejunum immediately at the anastomosis. Resection plus posterior polyanastomosis was done. The patient died on August 7.

Pathologic Examination.—The specimen consisted of a section of the intestinal tract measuring, when laid open, 15 by 10 by 1 cm. The serosa was thick and

fibrous. The muscle coats were distinct and well developed. The mucosa was edematous and hyperemic. The stoma of the enterostomy was red and smooth. There was little or no evidence of inflammation or ulceration. Microscopic examination of sections through the edge of the stoma showed chronic inflammation of the muscularis and some atypical glandular proliferation at the stoma. There was no frank evidence of neoplasm.

The clinical impression in this case was that the lesion was a jejunal ulcer. Although the roentgenologist stated that it was difficult to differentiate between a jejunal ulcer and a postoperative pocket, he concluded that a fleck was present. Gastroscopy did not permit good visualization of the jejunum but did reveal frank hemorrhagic gastritis. A thickened area was found at operation which was assumed to be the clinically diagnosed jejunal ulcer. Pathologic examination proved that no ulcer was present but that gastritis could be demonstrated microscopically. This case illustrates that roentgenographic examination, examination of a gross specimen or a gastroscopic examination is not infallible. The gastroscopic findings, however, were the most accurate in that they showed the presence of the inflammatory changes which were seen microscopically. It is, of course, possible that an ulcer in the lower part of the jejunum was overlooked at operation; this is not probable, however. Unfortunately autopsy could not be carried out.

CASE 19.—*History.*—A 45 year old patient, seen on Nov. 29, 1934, had an uncomplicated duodenal ulcer in March 1932. In December 1932 a posterior gastro-enterostomy was done because of considerable stenosis. A recurrence of epigastric distress developed in June 1934. This continued until two weeks previous to admission, at which time the patient noticed considerable epigastric distention. Five days before admission a severe cramplike pain developed across the epigastrium. Since that time the pain had recurred daily and had also been present at night.

Physical Examination.—No significant findings were noted.

Laboratory Examination.—The gastric fluid showed: free acid, 36, and total acid, 60. The stools gave a 1 to a 4 plus reaction to the benzidine test for blood.

Roentgen Examination (November 28).—The roentgen picture was negative for gastrojejunal ulcer.

Gastroscopy (November 22).—The interior of the jejunum was well seen; the complete circumference of the artificial opening could be observed. No ulcer and no peristalsis was observed. The folds were delicate, but the areas between the folds showed a grayish color. The diagnosis on the basis of the gastroscopic picture was rigidity of the artificial opening between the stomach and the jejunum and superficial gastritis.

Operation (December 13).—There was no stenosis of the duodenum. The gastro-enterostomy stoma was closed, and two large jejunual ulcers were excised. One of them was found to be directly in the suture line and had perforated the jejunum, its base lying against the transverse colon. The other ulcer lay about 2 inches (5 cm.) distal to the opening into the jejunum. The stoma of the previous gastro-enterostomy was closed.

Pathologic Examination.—There was a rather marked inflammatory reaction, with diffuse and focal round cell infiltration, and occasional areas of leukocytes were seen. No ulcer was found.

The patient was discharged, but in February pains again developed in the right side of the abdomen. These were most severe at night.

Roentgen Examination.—The roentgenogram of the stomach was normal. There were multiple diverticula of the sigmoid colon.

Second Gastroscopy.—In the pyloric region an opening was seen which was probably the pylorus itself. No other opening was seen. There was a circumscribed area on the surface of the posterior wall which contained thick layers of mucus. The other portions of the mucous membrane were normal. The patient was placed on medical management for ulcer on March 20, 1935, and felt "like a different person."

The explanation of this case is not entirely satisfactory. Two and a half years after gastro-enterostomy for duodenal stenosis, new pains developed. An ulcer could not be demonstrated by roentgen examination or gastroscopy or later by the pathologic studies. However, the surgeon who closed the gastro-enterostomy stoma found two large ulcers. Microscopic examination confirmed the gastroscopic diagnosis of chronic gastritis. Later, symptoms again appeared which were thought by the clinician to be of psychic origin and which completely disappeared on treatment for ulcer. We should probably assume that the observation of the surgeon was correct, in which case we have demonstrated a fact which is well known, namely, that jejunal ulcers may be overlooked by both roentgenographic and gastroscopic examinations.

CASE 20.—*History.*—A patient of Drs. L. Meyer and Necheles, at the Michael Reese Hospital, was seen on May 27, 1935. He had undergone resection for gastric ulcer one year before and at the present admission gave a typical history of ulcer.

Gastroscopy.—In spite of a marked kyphosis, the examination was carried out without difficulty. The jejunum was seen for an extent of about 5 cm. The Kerkring folds were seen so well that the presence of a deep jejunal ulcer could be ruled out in this case. The margin of the opening lying toward the greater curvature was somewhat swollen, and at the side of the anterior wall a small, shallow, yellow erosion was observed. The parts of the stomach lying above the opening showed marked changes consisting of red spots and layers of mucus. The mucous membrane was sensitive in this region. The diagnosis on the basis of the gastroscopic findings was postoperative stomach and severe gastritis of the mucous membrane with a superficial erosion of the artificial opening. The jejunum was fully visualized.

Operation (Dr. Strauss).—No lesion could be demonstrated from the outside.

The patient had previously had a gastric resection done, after which severe gastritis with marginal erosion developed. The symptoms were typical of a recurrent or jejunal ulcer. It is easy to understand that the surgeon could find nothing from the outside, since gastroscopy

showed no ulcer to be present. Another resection certainly would have brought no relief to the patient. We should probably have presented this case in our next group since the stomach was not opened, i. e., those cases without biopsic control. We wish to point out, however, that the findings of the surgeon agreed with those of the gastroscopist in that no real ulcer was present.

POSTOPERATIVE STOMACH WITHOUT BIOPSIC CONTROL

The cases of postoperative stomach without biopsic control offer so many interesting observations that they should not be omitted. However, we shall present them as briefly as possible in the following rough classification: (1) normal or fairly normal mucosa, (2) slightly abnormal mucosa, (3) recurrent ulcer, (4) jejunal ulcer, (5) simple erosions, (6) jejunal ulcer plus silk sutures, (7) silk sutures plus gastritis, (8) chronic gastritis, (9) gastritis plus perigastritis, (10) gastritis resulting from high voltage roentgen therapy and (11) unsatisfactorily explained cases.

Normal or Fairly Normal Mucosa.—CASE 21.—*History.*—A 40 year old woman had indefinite distress and pains and gas in the abdomen. She had formerly had a gastro-enterostomy done.

Physical Examination.—Abdominal scars and a large ventral hernia were noted.

Roentgen Examination.—The picture revealed a normal stomach.

Gastroscopy.—With the gastroscope introduced deeply, a mouthlike opening was seen in which circular folds appeared on its mucous membrane. When the instrument was withdrawn somewhat, the antrum appeared so that it was possible to observe the antrum, pylorus and artificial opening in the same visual field. The artificial opening contracted rapidly and closed completely (fig. 12) every fifth or sixth second. Its activity was entirely different from that of the pylorus. The antrum was limited by a marked musculus sphincter antri which was cordlike, whereas the lips of the artificial stoma were smooth, the upper one of which passed gradually into the mucous membrane of the posterior wall. Above the angulus thick gray mucus was seen lying between the folds of the greater curvature and the anterior wall; the upper parts of the stomach were normal. The diagnosis on the basis of the gastroscopic findings was rhythmic contractions of the gastro-enterostomy stoma in the posterior wall of the antrum of a type different from the contractions of the pylorus and slight superficial gastritis of the lower portions of the body of the stomach. (See *G* in the color plate.)

If one observes the postoperative stomach of a patient who has no complaints one usually sees an artificial opening with a pylorus-like rhythm. In this case the opening had its own peculiar rhythm of a type which we have never seen before. It is possible that this atypical action was responsible for the development of the superficial gastritis, which was slight. The patient's symptoms were probably caused by the ventral hernia with its adhesions plus a certain amount of apprehension.

Case 22.—*History.*—A patient of Drs. J. Meyer and Necheles, at the Michael Reese Hospital, had undergone resection of the stomach for gastric ulcer.

Gastroscopy.—A shallow cavity was seen at the end of the stomach. A dark hole which contracted rhythmically was observed at the end of this cavity. The edges of this hole were covered with some mucus, but there was no ulcer. All other parts of the stomach were normal. The diagnosis on the basis of the gastroscopic findings was rhythmic action of the artificial opening between the stomach and the intestine, but no ulcer or gastritis.

This case demonstrates that even in a stomach which has been resected it is possible that a rhythmic pylorus-like action may develop in the artificial opening, although it is so rare that this is the first time it has been described. The important observation is that no gastritis was present, which tends to confirm our belief that postoperative gastritis is the result of improper functioning of the artificial opening.

Fig. 12 (case 21).—Gastroscopic picture of a gastro-enterostomy stoma with functional adaptation. The same stoma is pictured in *G* of the color plate, but there the objective of the gastroscope was turned toward the posterior wall, and therefore only the gastro-enterostomy opening is seen. In this figure the pylorus and the gastro-enterostomy stoma are seen in the same visual field; the pylorus is open, and the stoma is closed with a stellar formation. A small portion of the pylorus was hidden behind the angulus. The greatest portion of the antrum is visible. It is evident that the gastro-enterostomy stoma lies close to the antrum.

Case 23.—*History.*—A patient of Drs. J. Meyer and Necheles, at the Michael Reese Hospital, had undergone resection of the stomach for gastric ulcer. The complaints at the present examination were suggestive of gastrojejunal ulcer.

Gastroscopy.—A spasm was felt at the lower pole, which soon relaxed, allowing good visualization. The artificial opening was seen; three fourths of its margin was quite smooth, but the remaining quarter, which lay toward the greater curvature, was nodular. The intestine with the Kerkring folds was well seen. Both loops were observed; they were separated by a definite ridge. The anterior loop showed peristaltic waves. There was no ulcer or inflammation in or near the jejunum. After the examination the patient appeared to be very nervous and complained of pain in her stomach. The diagnosis on the basis of the gastroscopic findings was no jejunal ulcer, slight gastritis in the uppermost parts of the stomach and tonic spasms of the stomach.

In spite of the fact that this opening was not entirely pylorus-like in its action, there was no severe inflammation and no ulceration. The spasm, together with the patient's general nervous temperament, seems to indicate that a psychoneurosis associated with painful spasms was responsible for her symptoms.

Slightly Abnormal Mucosa.—CASE 24.—*History.*—A 26 year old woman, seen on July 6, 1934, had had frequent hemorrhages from 1925 to 1935. Gastro-enterostomy was done in August 1928, and in October 1928 the stoma was closed.

Roentgen Examination (November 20).—The picture was that of a duodenal ulcer and an old operative defect of the stomach (distortion of the folds).

First Gastroscopy (November 1).—The antrum was irregular in form. Radial folds were seen running toward the pylorus, which remained hidden under an irregularly bent fold. The fold was probably the angulus distorted by operation. Two small nodules were present on the anterior wall. The diagnosis on the basis of the gastroscopic findings was scar formation in a postoperative stomach and residuals of gastritis.

Second Gastroscopy (Feb. 1, 1935).—The picture was similar to that seen at the first gastroscopy.

The frequent hemorrhages were probably due to a duodenal ulcer. Four years previously the gastro-enterostomy stoma was closed. The stomach showed distortion caused by this operation and residuals of gastritis. This case demonstrates the good results often obtained by eliminating a gastro-enterostomy stoma.

Recurrent Ulcers.—CASE 25.—*History.*—A woman aged 32, seen on Jan. 19, 1935, had undergone gastro-enterostomy in 1930 for gastric ulcer. She felt well until March 1, 1934, when she complained of "gas around the heart" and cramplike pains, which usually settled in the upper left abdominal quadrant.

Physical Examination.—There was tenderness in the epigastrium.

First Roentgen Examination (January 19).—There was at least one, and probably two, penetrating ulcers on the lesser curvature immediately above the angulus. An old duodenal deformity was seen. The gastro-enterostomy stoma was patent. The possibility of an associated mild gastritis could not be ruled out.

First Gastroscopy.—The entire stomach, including the lesser curvature of the antrum, was seen. The pylorus functioned normally. At the depth of the angulus behind a fold of the posterior wall was seen the artificial opening of a gastro-enterostomy which showed activity like that of a normal pylorus; e. g., when it closed it formed the well known stellar formation, and when it was open it showed a round hole. Since both openings could not be seen in the same visual field, it could not be observed whether the rhythm was the same. The mucous membrane of all these parts was normal. Above the angulus, along the lesser curvature yellowish gray, irregular ulcerations were seen. Most of them did not give the impression of being deep; the crater-like appearance was missing, and yet they did not resemble the ulcerations of typical gastritis ulcerosa; six of these ulcers were counted. The surrounding mucous membrane did not show marked inflammation, although the edges of the ulcers were somewhat reddened. The diagnosis on the basis of the gastroscopic findings was numerous shallow ulcers of the lesser curvature and pylorus-like activity of the gastro-enterostomy opening.

Second Gastroscopy (February 25).—All ulcerations had completely disappeared. On the anterior wall was seen a small dark brown erosion (which had the typical appearance of the so-called "hemorrhagic erosion"). In the highest parts of the lesser curvature red spots were seen which were not sharply limited. In the surrounding pale area a big brown pigment spot was seen. The diagnosis was one small hemorrhagic erosion in the anterior wall, a large pigment spot and completely healed ulcerations.

Second Roentgen Examination (February 28).—The penetrating ulcer formerly present at the lesser curvature of the stomach had disappeared.

Third Gastroscopy (May 16).—The Kerkring folds and the whole margin of the stoma were seen. Two small mucosal hemorrhages were present, but the hemorrhagic erosion had disappeared.

Fourth Gastroscopy (July 18).—The gastro-enterostomy opening was working rhythmically. There were no erosions, no hemorrhages and no pigment spots.

Because of the frequent gastroscopic observations, this case is particularly interesting. The patient did not have a history that was typically suggestive of ulcer. Roentgen examination revealed the old gastro-enterostomy stoma and one or two ulcers of the lesser curvature. Gastroscopic examination demonstrated that the gastro-enterostomy opening worked rhythmically and in a pylorus-like manner. It demonstrated further the presence of six ulcers of the lesser curvature with no gastritis. The ulcerations all healed completely in one month under medical management. The typical hemorrhagic erosion in an otherwise normal stomach which was seen is a frequent finding in "ulcer stomachs." We feel certain that Konjetzny [15] would not have denied their existence if he had performed gastroscopy on his patients. Some months later the stomach was normal except for the presence of two small hemorrhages; these are also frequently seen in "ulcer stomachs." They may change into pigment spots or into hemorrhagic erosions and finally into ulcer. The excellent result of medical treatment in this case was remarkable. The fact that there was no gastritis present was probably because of the rhythmic pylorus-like function of the gastro-enterostomy opening.

Jejunal Ulcer.—CASE 26.—*History.*—A 58 year old man, seen on Oct. 24, 1934, had a condition which was diagnosed as duodenal ulcer with obstruction on Feb. 12, 1929. An anterior gastro-enterostomy was done. On July 26, 1932, a second operation was done, at which numerous bleeding jejunal ulcers were found. A part of the jejunum was excised, and the jejunum and stomach were closed. Ulcerated hemorrhagic areas were observed in the intestine.

On Aug. 1, 1933, duodenal ulcer with a high grade of stenosis was again found. Partial gastrectomy was done on August 4. Two ulcers of the stomach were observed. On Oct. 24, 1934, the patient again presented himself, complaining of having had severe pain daily in the left abdominal quadrant.

Physical Examination.—Tenderness was elicited in the left upper quadrant.

15. Konjetzny, G. E.: Die entzündliche Grundlage der typischen Geschwürsbildung im Magen und Duodenum, Berlin, Julius Springer, 1930.

First Roentgen Examination (Oct. 26, 1934).—A large recurrent ulcer was seen at the lesser curvature of the gastric stump.

First Gastroscopy (October 27).—The examination was unsatisfactory. The artificial opening was seen, and no peristaltic movements were observed. There was a small area of superficial gastritis.

Second Gastroscopy (December 7).—The picture had changed completely. The new opening was seen, and on its upper edge there was a deep ulcerated crater the floor of which was covered with whitish yellow layers and the edges of which were extremely reddened and hemorrhagic. The folds of the mucous membrane as well as the valleys between them were covered with yellowish layers of pus. The mucous membrane surrounding the opening was exceedingly swollen and reddened. In the upper parts of the stomach the folds became thin, their ridges were red, and the valleys between them were grayish red. The diagnosis on the basis of the gastroscopic findings was a deep marginal ulcer and suppurative hypertrophic gastritis surrounding the opening. (See *H* in the color plate.)

Third Gastroscopy (December 13).—The ulcer was seen better than at the previous examination. It was quite evident that it was situated in the wall of the jejunum itself. Its edges were reddened, and whitish layers of mucus were seen on one side of the edge. The surrounding mucous membrane, however, was no longer as extremely swollen as it was during the previous examination. The diagnosis on the basis of the gastroscopic findings was jejunal ulcer and improvement of the associated inflammation.

Second Roentgen Examination (December 29).—An ulcer at the region of the gastric anastomosis could no longer be demonstrated.

Fourth Gastroscopy.—The examination was unsatisfactory. There was so much mucus and secretion present that the jejunum was not seen.

Fifth Gastroscopy (Jan. 30, 1935).—The jejunum and ulcer were seen instantly with the straight tipped instrument. The ulcer was shallow and yellowish, and the surrounding area was not swollen. There was no secretion present. The diagnosis on the basis of the gastroscopic findings was a healing jejunal ulcer and no gastritis or jejunitis.

Sixth Gastroscopy (March 5).—The opening appeared to be narrower. The border could be well seen; there was a small grayish scar, but no ulcer was present nor was any inflammation seen. The diagnosis was apparent healing of the ulcer and gastritis.

Seventh Gastroscopy.—Visualization was excellent. Slight superficial gastritis was seen, and the ulcer was healed.

Eighth Gastroscopy (May 2).—The first 2 to 3 cm. of the jejunum was well seen. No ulcer or inflammation was present. On the side opposite where the previously seen jejunal ulcer had been a small but definite yellowish patch was observed which remained constant during the entire examination. The mucous membrane of the stomach was essentially normal. The yellow spot might have represented the beginning of a new jejunal ulcer.

Ninth Gastroscopy (July 5).—The picture was similar to that seen on the last examination. There was considerable secretion in the jejunum, and the mucosa was somewhat inflamed, but there was no ulceration either in the stomach or in the jejunum.

Tenth Gastroscopy.—The jejunum appeared to be normal. The inflammatory changes were much improved over those seen at the last examination. There

seemed to be a cobble-stone appearance of the mucous membrane of the fornix. No ulcer was seen. The jejunum was well visualized, and there was a small residuum of inflammation.

After the patient underwent several operations a jejunal ulcer developed. It was possible to watch the progress by a series of ten gastroscopies in spite of the fact that he was stout and had an unusually sensitive gastric mucosa. The excellent visualization of the ulcer in the interior of the jejunum was unusual. The healing of the ulcer under medical management (Winkelstein tube) was most interesting to watch. Gastroscopy in this particular case was definitely superior to roentgen examination.

Simple Erosion.—These lesions have been seen in two preceding cases to which we refer the reader. In case 19 an erosion without gastritis was observed which, of course, was not seen in the gross specimen after resection. Later gastroscopies were done in this case, which demonstrated similar erosions. Finally, a severe inflammation developed.

A typical hemorrhagic erosion was observed in case 25 in a noninflamed mucosa after the healing of six true ulcers of the lesser curvature. This erosion healed without consequence.

Jejunal Ulcer and Silk Sutures.—In case 14 the large silk sutures were found in the gross specimen hanging into the cavity of the stomach. It might be assumed that the presence of these sutures was of consequence in the development of the jejunal ulcer. This, however, cannot be proved.

Silk Sutures Plus Gastritis.—The following four gastroscopic examinations which have not yet been cited were done in case 1, after resection of a benign obstructing ulcer of the duodenum.

Second Gastroscopy (Sept. 21, 1934).—There was a high wall seen which separated the body of the stomach from a round black hole, which apparently was the artificial opening. Toward the anterior side there was a mushroom-like elevation, the middle of which was drawn in and appeared to be puckered. A black silk thread was seen which had not cut through the mucous membrane.

Third Gastroscopy (November 1).—All findings of a superficial gastritis were observed. Many red spots were seen, and large collections of mucus lay between the folds. The artificial opening was open and rigid. Silk threads were well seen.

Fourth Gastroscopy.—It was possible to penetrate into the jejunum with the gastroscope, so that a long portion of it was well observed; it was found to be normal. The wall of the opening was thick and somewhat reddened. Above this point in the anterior wall there were two sutures (silk threads) which were hanging partially free into the interior of the stomach. The areas surrounding them were spotted and covered with mucus.

We were unable to follow the patient further because he had to return to his home in Canada, but it is important to note that gastritis was observed surrounding the sutures.

CASE 27.—*History.*—A patient of Dr. J. Meyer, at the Michael Reese Hospital, had a gastro-enterostomy done for gastric ulcer on Aug. 5, 1935. He later complained of pain in the left upper abdominal quadrant.

Gastroscopy.—The antrum was normal. There was a black silk suture in the lowest part of the posterior wall which had cut through the mucous membrane at the side of a fold, behind which probably lay the gastro-enterostomy stoma. In the upper parts of the posterior wall some small nodules were observed. Just below the cardia there was a network of blue blood vessels lying in an area having a greenish gray color. One hemorrhagic spot was observed between the nodular part and the discolored area. The diagnosis on the basis of the gastroscopic picture was the presence of a black silk suture which had cut through the mucous membrane and hypertrophic and atrophic gastritis of the upper parts of the body.

These silk sutures which have penetrated the mucous membrane cause a severe gastritis, often with erosions, and although it seemed possible that those in case 1 may have been expelled eventually, they usually have to be removed surgically. When this is done the patient often becomes entirely free from symptoms. For these reasons we believe that the presence of sutures which have penetrated the mucous membrane constitutes a definite indication for operation.

Chronic Gastritis.—In 1922 one of us (R. S.) [14] described the frequency, importance and seriousness of chronic gastritis in the postoperative stomach. Following this, all gastroscopists and later many surgeons and internists agreed that chronic gastritis is mainly responsible for the distress so frequently found after operations for gastric lesions. Since chronic gastritis seems to develop much more frequently in those cases in which the action of the artificial opening is not pylorus-like, we may assume that the intestinal juice flowing into the stomach through this patent opening is in some way responsible for the origin of most of these severe gastritides. It may be dependent on a chemical or a bacteriologic influence, or it may be the result of a combination of these factors. It is true that the gastritis may sometimes be present before the operation, but by observations cited in this paper it becomes evident that there are cases in which there was no gastritis previous to operation (case 19) and in which it developed to a severe extent postoperatively. Evidence seems to indicate further that this type of gastritis may be healed by eliminating a gastro-enterostomy stoma (case 16). The classification of chronic gastritis into three divisions (superficial, hypertrophic and atrophic) does not hold in the postoperative stomach. Henning has tried to sustain this classification, but since all combinations are seen and the prognosis is always the same, e. g., the gastritis heals only rarely and then usually in cases in which the gastro-enterostomy opening has been eliminated, we shall not attempt to do so. There is no great difference between the pictures of the gastritis found in the stomach which has been resected and in the stomach in which a gastro-enterostomy has been done. We find, however, in a study of a great

number of cases, that the opening of the gastro-enterostomy more frequently shows the rhythmic pylorus-like action than does the opening of the stomach which has been resected. A gastro-enterostomy is advantageous in that the stoma can be closed if necessary.

Before presenting any additional cases, we shall refer to four cases already reported (cases 15, 16, 17 and 19). Case 15 presented symptoms which were typical of an ulcer after a gastro-enterostomy had been done. Gastroscopically and microscopically, a severe gastritis without ulcer was found. In case 16, in which a gastro-enterostomy was also done, frequent hemorrhages occurred. At gastroscopic examination no ulcer was found, but a severe gastritis was seen. At the following operation no ulcer was demonstrated and the old duodenal ulcer seemed to be healed; the gastro-enterostomy opening was closed. After this the gastritis healed.

In case 17 it was thought, on the basis of the clinical picture, that a jejunal ulcer developed after gastro-enterostomy. Gastroscopic and microscopic examinations showed the nonexistence of an ulcer and demonstrated the presence of severe gastritis.

In case 19 there was a shallow erosion with some hemorrhages but no gastritis. After resection severe gastritis developed.

CASE 28.—*History.*—A patient presented a history of ulcer since 1922. Gastro-enterostomy was done in 1922 and undone in 1928. Uncomplicated duodenal ulcer occurred in 1929; resection of the pylorus was performed in 1932 for the duodenal ulcer and in 1933 for a jejunal ulcer (pathologically confirmed). The patient was seen on April 13, 1935, because of recurring epigastric distress. He had suffered from pain in the right shoulder for the past four months. The distress had been present since the last operation.

Physical Examination.—A number of abdominal scars were present. No tenderness was elicited, and the liver was just palpable.

Roentgen Examination.—There was no definite evidence of fleck in the stomach. The patient localized his tenderness to the point of the anastomosis. The appearance of the rugous folds was suggestive, but not conclusive, of gastritis.

Laboratory Examination.—The gastric contents showed free acid up to 122 by the histamine test.

First Gastroscopy (October 5).—The opening into the jejunum was well seen; its mucous membrane and that of surrounding areas of the stomach was reddened and showed many irregular nodules and hemorrhages. These changes were also present in the upper parts of the stomach, but to a lesser extent. The diagnosis was hypertrophic hemorrhagic gastritis in a postoperative stomach.

Second Gastroscopy (Jan. 29, 1935).—The artificial opening was completely rigid. There were no great changes from the previous examination. The secretion of mucus and pus was more extensive than before, especially in the jejunum where all the valleys between the Kerkring folds were covered with a thick secretion. The mucous membrane of the stomach was extremely red.

Three more gastroscopic examinations were carried out on the following dates: March 13, April 20 and July 15. Each of these demonstrated the picture that

has already been described. The patient was given the Winkelstein treatment, and although his distress seemed somewhat less at times it always recurred with its usual intensity.

A gastric resection was done on this patient after several previous operations had been carried out. An ulcer was found at the time of resection, but no gastritis was present. After operation marked gastritis developed, which was well observed through a series of six gastroscopies. A recurrent ulcer could not be demonstrated either by roentgen examination or by gastroscopy. In contrast to case 26, in which a recurrent ulcer was present, in this case there was no response to the Winkelstein treatment. The case illustrates the poor prognosis offered by gastritis in a stomach after resection.

CASE 29.—*History.*—A 60 year old man, who had undergone resection of the stomach for carcinoma in 1930, was seen on Oct. 21, 1935. For the past two to three months he had complained of pain, nausea and anorexia.

Roentgen Examinations (Six).—There was no evidence of obstruction. Gross dilatation of the intestinal coils was noted in the small intestine.

Gastroscopy (November 11).—The artificial opening was easily seen. In the upper parts of the stomach some small patches of thick mucus were observed. Just below the cardia the mucous membrane became thin, and two large blood vessels were seen. The diagnosis on the basis of the gastroscopic findings was atrophic and superficial gastritis of a slight degree in the upper parts of the stomach. The patient's condition might be expected to run a favorable course.

The atrophic form of gastritis is not frequently seen in a stomach after resection. In this case it may have been present before resection, accompanying the carcinoma, which was removed five years previously. Here are seen the good results which may be expected from early surgical removal of a carcinoma of the stomach.

CASE 30.—*History.*—A 56 year old man had undergone subtotal resection for peptic ulcer in 1924 at the Cook County Hospital. He had had an intermittent burning type of distress in the epigastrium ever since. This was not relieved by the ingestion of food and little by the ingestion of alkalis. During the past two years it had become worse. The patient was seen on Aug. 19, 1934.

Physical Examination.—There were epigastric tenderness and a systolic murmur at the base of the heart.

Laboratory Examination—The gastric fluid obtained after an Ewald test meal showed: free acid, 0, and total acid, 16.

Roentgen Examination.—Two gastrojejunal ulcers were probably present with marked thickening of the gastric mucosa.

Gastroscopy (August 22).—The jejunum and its folds were easily seen. A brown silk thread was noted near the operative opening which had not cut through the mucous membrane. Hypertrophic changes were seen on the anterior wall in the superior parts of the stomach; there were also small hemorrhages and some edema. At one point there was a distinct erosion, which was very shallow and which had a whitish base. Other parts of the stomach were normal. The diagnosis was hemorrhagic hypertrophic gastritis after resection.

The distress of which the patient complained was undoubtedly due to the marked hypertrophic gastritis which was found to be present.

The following two cases of gastritis after resection show definite signs of perigastritis. The first of them was further complicated by gastric hemorrhage.

CASE 31.—*History.*—A 37 year old man, seen on May 10, 1935, gave a history of epigastric distress of long duration which was suggestive of ulcer. Partial gastrectomy was performed in November 1932, after which a hemorrhage occurred. The patient was again operated on on Feb. 3, 1935, for jejunal ulcer. He stated that he had recently had another hemorrhage.

Physical Examination.—No significant findings other than numerous abdominal scars were noted.

Roentgen Examination (June 17).—There was no definite evidence of an ulcer crater.

Laboratory Examination.—The histamine test of the gastric fluid showed free acid up to 72.

First Gastroscopy (May 11).—The examination was unsatisfactory because of the poor cooperation of the patient. Hypertrophic gastritis was seen.

Second Gastroscopy (May 14).—The patient was quiet, and the anterior margin of the artificial opening was well seen. Beyond it the mucous membrane of the stomach prevented visualization of the posterior margin and the jejunum itself. The mucous membrane of the margin and that surrounding it was red and swollen; creases were seen. There were marked defects in the epithelium which were so small that they could scarcely be called ulcerations. Spots of mucus were observed in the lower parts of the stomach. The diagnosis was postoperative distortion of the entire stomach (Moutier would call this "perigastritis" and would probably be correct in doing so) and severe erosive hypertrophic gastritis. A jejunal ulcer could not be ruled out; however, the small erosions could account for the bleeding.

Third Gastroscopy (July 15).—The examination was unsatisfactory since the wide-angled instrument was not available.

Fourth Gastroscopy.—This gastroscopy was done at the patient's request. The entire stomach was well seen. The artificial opening was observed, but a small part of its edge could not be seen. The mucous membrane of the jejunum appeared to be normal. The edge of the stomach was markedly reddened and swollen; the folds in the body were serpiginous. A solitary reddened node was seen in the midportion of the greater curvature. Thick grayish white mucus was observed between the folds just below the cardia. There was improvement in the hypertrophic gastritis.

The patient presented a history of ulcer of long duration for which partial gastric resection was done. Later, a complete examination was done after an extensive hemorrhage. No ulcer was found, but gastroscopy revealed severe erosive hypertrophic gastritis. Although an ulcer could not be ruled out with certainty, it is most likely that the hemorrhage was caused by the erosions. There was evidence of perigastritis. The gastritis improved but did not disappear completely.

CASE 32.—*History.*—A 64 year old man, seen on Nov. 2, 1935, two years previously had amebic dysentery. About a year before a gastric resection was done for peptic ulcer. The present complaint was anorexia and loss of weight. The patient wished a complete check to rule out carcinoma.

Laboratory Examination.—The gastric fluid after an Ewald test meal showed: free acid, 0, and total acid, 35. The stools were normal.

Roentgen Examination.—There was no evidence of a lesion in the gastric resection; the stoma functioned well.

Gastroscopy.—Orientation was difficult because of the extreme postoperative distortion. In the lowest part of the stomach there were many serpiginous folds between which many nodules were seen. In the area probably corresponding to the old suture line a hemorrhagic node and thick layers of mucus were seen. At a higher level the folds of the greater curvature were seen, and toward the anterior wall appeared the oval dark opening of the gastro-enterostomy. Dr. Renshaw thought that he saw circular folds within this opening, but they could be seen only at the moment of the height of inspiration. In the upper parts of the stomach one hemorrhagic node and many smaller nodules were obeserved. The diagnosis was greatly distorted postoperative stomach; hemorrhagic, nodular, hypertrophic gastritis, and a patent artificial opening.

It is doubtful whether this patient ever had an actual ulcer. A large resection was done, however, and when he presented himself at the clinics he complained only of anorexia and loss of weight. The clinical examination gave negative results. Gastroscopy revealed hypertrophic gastritis and an extremely distorted stomach. Distortion is usually seen better by roentgen examination than by gastroscopy. In accord with Moutier, we did not hesitate to assume that perigastritic adhesions, which apparently did not bother the patient, were present.

CASE 33.—*History.*—A 39 year old man, referred by Dr. B. Goldberg on Nov. 6, 1935, had a gastro-enterostomy performed seven years before; the surgeon found a duodenal ulcer. The patient's complaint at the time of the present examination was epigastric pain associated with cramps. He was relieved by eating. The region around the gastro-enterostomy stoma was not painful on manipulation before the fluoroscope.

Gastroscopy.—The antrum and the action of the pylorus were well seen. There was a marked cordlike musculus sphincter antri over the anterior wall. Toward the greater curvature and below this suture line the gastro-enterostomy opening was seen. It was patent but did not contract. As the instrument was withdrawn large nodes were seen on the posterior wall and some smaller ones on the anterior wall. The folds of the posterior wall were segmented, and a dark crease could be seen crossing one of them. A solitary node was seen on the lesser curvature.

The diagnosis on the basis of the gastroscopic findings was marked hypertrophic gastritis of the body of the stomach.

This case illustrates the usual sequela of a patient with a nonrhythmically working gastro-enterostomy opening, i. e., marked hypertrophic gastritis.

CASE 34.—*History.*—A patient, referred by Dr. Federman, had had a gastro-enterostomy performed because of benign duodenal obstruction. His complaint at the time of the present examination was epigastric pain.

Gastroscopy.—The entire stomach was well seen. The greater curvature of the antrum showed large, tortuous, thickened folds, with some creases and hyperemic spots. There was no ulcer or inflammation in the area of the gastro-enterostomy stoma. The upper parts of the stomach were normal. A diagnosis of severe gastritis of the greater curvature of the antrum was made.

As in case 34, gastroscopy showed severe hypertrophic gastritis in a patient with a patent gastro-enterostomy stoma. Since the inflammation was restricted to the greater curvature in this case, it is possible that it may have originally been caused by the obstruction.

CASE 35.—*History.*—A 43 year old man had had a gastro-enterostomy performed five years before the present examination. His present complaint was diarrhea persisting for five weeks, belching and epigastric distress. He stated that he felt better if he did not eat.

Physical Examination.—There were no significant findings.

Laboratory Examination.—The gastric fluid after an Ewald test meal showed: free acid, a trace, and total acid, 60. The stools gave a 1 plus reaction to the benzidine test for blood.

Roentgen Examination.—The gastro-enterostomy stoma was patent. No jejunal ulcer was demonstrated.

Gastroscopy.—The pylorus was well seen; across the greater curvature of the antrum ran a solid narrow fold. On the posterior wall near the pylorus there was an opening through which the interior of the intestine was easily seen. The new opening did not close during the peristalsis. The lower border of the gastro-enterostomy stoma showed definitely reddened areas, as did also the folds of the anterior wall. There were many small nodules in the mucous membrane of the lesser curvature. The diagnosis was absence of rhythmic action of the gastro-enterostomy opening and slight chronic gastritis (superficial plus hypertrophic) of most portions of the stomach, but most marked in the area surrounding the opening.

Although the gastro-enterostomy stoma was situated in that part of the stomach most favorable for the development of the pylorus-like rhythm, none was present. Chronic gastritis of all portions of the stomach was present, which improved to a marked extent on dietary management. However, our past experience has shown that exacerbations frequently occur.

CASE 36.—*History.*—A 59 year old man, seen on April 23, 1935, in 1930 had a cholecystectomy and gastro-enterostomy done (duodenal ulcer found). He was well for the following three years, but then attacks of dull pain developed in the right upper abdominal quadrant.

Physical Examination.—No significant findings were noted.

Roentgen Examination (April 25).—The gastro-enterostomy stoma was patent. There was localized tenderness at the region of the stoma.

Gastroscopy (April 25).—The entire stomach seemed to be distorted, and the orientation was difficult. The pylorus was not seen. A sickle-shaped fold was

seen on the posterior wall which probably covered the gastro-enterostomy stoma. Toward the cardia the mucous membrane was velvet-like and swollen and contained many crevices. Toward the lower pole of the stomach there were also rather superficial circumscript changes; thick stripes of grayish mucus were seen. The mucous lake in the fornix was more transparent than usual and did not present its usual color. The diagnosis was circumscribed gastritis surrounding what was probably the funnel-shaped artificial opening. There were probably adhesions in the region of the pylorus and of the gastro-enterostomy stoma.

Three years after gastro-enterostomy was done abdominal distress again developed. There was no evidence of a recurrent ulcer. Gastroscopy revealed a circumscribed hypertrophic gastritis around the stoma. Perigastritic adhesions were also undoubtedly present.

Case 37.—*History.*—A 39 year old woman, seen on Nov. 28, 1934, had intermittent attacks of abdominal distress for eight years. In 1927 she underwent an operation for ulcer. After this she complained of a dull pressure pain in the epigastrium, which was more intense after meals. One week before the present examination severe diarrhea developed, followed by nausea and vomiting.

Physical Examination.—No significant findings were noted.

Laboratory Examination.—The gastric fluid after an Ewald test meal showed: free acid, 0; total acid, 12, and lactic acid, 3 plus. The stools were negative for blood.

Gastroscopy.—The action of the pylorus was normal. Just above the antrum the mucous membrane of the posterior wall showed no folds and appeared to be edematous. At a slightly higher level the gastro-enterostomy stoma was seen; there was no peristaltic contraction, and the Kerkring folds of the jejunum were marked. Numerous hemorrhages were seen on one fold of the lesser curvature. The other parts of the stomach were normal. The diagnosis was absence of peristalsis of the gastro-enterostomy stoma in the posterior wall; superficial gastritis of the lower parts of the posterior wall, and hemorrhages in one fold of the lesser curvature.

Superficial edematous gastritis is a common finding in the postoperative stomach in which the artificial opening is rigid. The finding of lactic acid without obstruction is frequent in such cases.

Gastritis After High Voltage Roentgen Therapy.—It is well known that roentgen treatment after the manner of Coutard for neoplastic lesions of the mouth causes a severe inflammation of the mucous membrane which heals after a certain length of time. The same observation has been made by one of us (R. S., in association with Dr. Sielmann) in cases of gastric neoplasm in which several of the patients were treated by the method of Coutard with excellent results.[16] The patients had not previously been operated on, so one may conclude that undoubtedly the severe inflammation was caused by the roentgen therapy. The gastritis following the Coutard treatment in case 12 occurred

16. Schindler, R., in discussion on Sielmann, H.: Erfahrungen mit der Röntgentiefenbestrahlung nach Coutard bei malignen Neubildungen, München. med. Wchnschr. **78**:384 (Feb. 27) 1931.

after resection had been done, so that it is not known to what extent this influenced the gastritis, but undoubtedly the major cause was the high voltage roentgen therapy. Gastroscopy was carried out thirteen days after the first treatment. On inspection the new opening was seen to be slitlike and to open rarely; it functioned with a rhythm peculiar to itself (fig. 13). The border of the opening was grayish yellow, and the mucous membrane of almost the entire stomach was red and swollen. In the lesser curvature there were linear areas of mucosal hemorrhages. Thick layers of dirty grayish mucus were observed on the posterior wall, thus making it impossible to state with certainty whether metastatic lesions were present. There was an extremely severe gastritis of almost the entire stomach, which was most probably due to the Coutard method of high voltage roentgen therapy.

Unsatisfactorily Explained Cases.—In four cases (cases 38 to 41) only a part of the stomach was seen. For this reason we shall not report

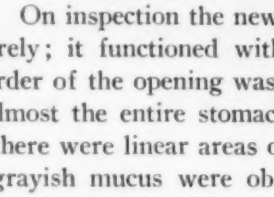


Fig. 13.—Gastroscopic picture of a slitlike stoma after gastric resection because of carcinoma of the pylorus. This stoma had a rhythmic function like a real pylorus. Pylorus-like action of an artificial stoma after resection has not yet been described.

these cases other than to state briefly in what respects they were not satisfactory. In the first of them a peculiar crater was observed a few cubic centimeters from the edge of the gastro-enterostomy opening. A definite interpretation of this crater could not be made. Gastroscopy in the second case was done in the presence of a large audience; this disturbed the patient. Although chronic gastritis was found, the gastro-enterostomy opening could not be seen with certainty. In the third case an erosive gastritis was demonstrated, but here again the artificial opening could not be seen; furthermore, an unusual protrusion of the antrum could not be explained satisfactorily. In the final case gastritis was also demonstrated but an artificial opening was not disclosed.

Conclusions.—Gastroscopy of the postoperative stomach is not easy, since the operation produces changes in the contour which makes orientation difficult. The results with the Schindler-Wolf flexible gastroscope,

however, are much more accurate than were those with the rigid instrument in which a maximum of only 50 per cent of artificial openings could be seen. In our series gastroscopy was performed on a total of thirty postoperative stomachs, on twelve after resection and on eighteen after gastro-enterostomy. The artificial opening was well observed in every stomach on which resection had been done. The gastro-enterostomy stoma was not demonstrated in three cases, or 10 per cent of all our postoperative cases. In two instances the opening was partially obscured by large folds.

Postoperative stomachs should be divided into two groups: those with a rhythmically working stoma and those with what we designate as a "patent stoma," by which we mean complete absence of activity at the opening, even though peristalsis in the intestine beyond the stoma is observed. The pylorus-like rhythm which develops in certain instances is an interesting and important phenomenon. Although it does not prevent the formation of recurrent ulcers, it does seem to prevent the development of chronic gastritis, which unfortunately is such a frequent postoperative occurrence and one which makes the operative result not wholly satisfactory. We observed this pylorus-like rhythm in four cases, twice in stomachs after resection and twice in stomachs after gastro-enterostomy. Functional adaptation thus took place in 13.3 per cent of all the postoperative stomachs which we observed, but we feel certain that it actually develops in a much higher percentage since gastritis does not develop, or at least infrequently, and hence the patient is not likely to present himself at the clinic. From the past experience of one of us (R. S.), we believe that this adaptation is probably more likely to occur after gastro-enterostomy than after resection, although we have no definite proof of this. At least gastro-enterostomy has the advantage that the stoma may be closed. More work should be done in respect to this entire problem by following the postoperative stomach gastroscopically.

It seems evident after gastroscopic study that the activity of the pylorus and the artificial stoma is not governed by a joint nervous center, since we have seen two cases in which both openings were observed in the same visual field and the rhythms were different. When the new stoma acquires this functional adaptation it presents the same stellar appearance when closed as has been described in respect to the pylorus. The higher centers may have some control over the pyloric activity, but the actual control of the rhythm probably lies in a center located in the wall of the stomach itself.

The patent opening often permits visualization of both loops of the intestine. The margin of the stoma may be stiff and even nodular as a result of operation; these nodes and postoperative pouches are frequent, and the nodes may be so large as to be mistaken for real tumors.

In our statistics of all gastroscopies done at the Billings Hospital we found 20 per cent of the stomachs normal, whereas among the thirty postoperative cases only three, or 10 per cent, appeared to be without significant lesions.

Recurrent ulcers were found in one case in which a rhythmically working gastro-enterostomy stoma was visualized. In this case six ulcers were found lying on the posterior wall close to the pylorus; only one of them was seen with certainty by roentgen examination. They all healed rapidly under medical management. No gastritis was observed at any time, but later small hemorrhages and one typical hemorrhagic erosion were seen. We believe such changes are frequently found in the ulcer type of stomach.

One jejunal ulcer was missed by gastroscopy; it was also not seen by roentgen examination. In one case the course of a jejunal ulcer was followed by a series of ten gastroscopic examinations; the ulcer was situated in the interior of the jejunum and was accompanied by severe gastrojejunitis. It healed completely under the Winkelstein method of treatment.

A large marginal ulcer of the stoma was seen in one case in which black silk threads were found hanging free into the cavity of the stoma. It was impossible to say to just what extent, if any, these threads were responsible for the development of the ulcer. We do know, however, that silk threads which cut through the mucous membrane after operation may cause trouble if they are not soon expelled. They first cause an inflammation; later erosions develop from the resulting friction. For these reasons they should be removed by operation.

All gastroscopists and many surgeons (Clairmont [13]) agree that chronic gastritis is the most frequent and important postoperative disease. It is true that in some cases gastritis may be present before the operation; our cases have shown, however (especially case 19), that gastritis does develop after operations on stomachs in which it was definitely not present before. Postoperative gastritis presents the most severe pictures that one sees of this condition, and since almost every case includes all types of this inflammation (superficial, hypertrophic and atrophic), we see no reason for attempting any descriptive classification. Thus we will add a fourth group to the three types already mentioned which we shall call simply "gastritis of the postoperative stomach." We do not know the exact etiologic factors which are responsible for the development of postoperative gastritis, although there is evidence that the constant reflux of intestinal juices may cause and maintain the inflammation. This is shown by the fact that in those cases in which there are no postoperative difficulties we generally found a rhythmically functioning stoma (one

exception, case 23), which by its closing prevents the reflux of intestinal contents. Of the four cases which we observed in which a rhythmically functioning stoma was present, in none was gastritis observed except one which was seen after high voltage roentgen therapy by the Coutard method, which always produces severe gastritis. In two of the cases the stomach was normal; in the remaining one there was no gastritis but recurrent ulcers, which later completely healed. There is no doubt that patients suffer from this postoperative gastritis at least as much as they do from jejunal or recurrent ulcers; furthermore, gastritis occurs much more frequently (sixteen cases in our material). In addition to this, all observers agree that severe hemorrhages frequently result from this condition; this was observed twice in our cases. The treatment of gastritis is difficult, and for this reason the important task of the surgeon is to prevent its development. We have already stated that we believe that gastritis rarely develops if a rhythmically functioning stoma results. In cases of resection we do not know how the surgeon can favor the development of this functional adaptation, but we agree with Moutier that if the gastro-enterostomy stoma is made in the posterior wall close to the pylorus it is much more likely to take place. We will add that the opening should be made not only near the pylorus, on the posterior wall, but also close to the greater curvature. Further investigations must be made concerning this important question, because only by the prevention of chronic gastritis can operations for ulcer be completely satisfactory. The rôle which perigastritic adhesions play as a cause of postoperative distress has not been explained by our observations. They were encountered three times, but in only one case was there not also an associated gastritis. It is questionable in this case whether the adhesions were responsible for the symptoms, although it is not impossible. If it seems quite certain that they are the cause of distress, it seems probable that they should be removed surgically, although we know that this operation is generally of no great value.

In this series of cases we did not observe any instances of carcinoma at the gastro-enterostomy stoma.

SUMMARY

This paper is based on the findings of seventy-eight gastroscopic examinations carried out on forty-one patients. Thirteen of the forty-one patients underwent gastroscopy preceding operation and two of the thirteen again after operation. The remaining twenty-eight patients were all examined postoperatively; seven of this group again underwent operation after gastroscopy, thus giving us a biopsic control in twenty cases.

Although the gastroscopic diagnosis was confirmed in every case in which we had biopsic control (twenty cases), it must nevertheless be admitted that negative findings in gastroscopy are not entirely conclusive.

The differential diagnosis between benign and malignant ulceration is not difficult to determine after a gastroscopic examination by one trained in this field; even in the very early stage biopsy is not necessary. The differentiation between benign and malignant obstruction is more difficult but also possible. Gastroscopic diagnosis was proved to be correct in each instance (seven cases). The gastroscopic picture is more characteristic than is that of the gross specimen because of the presence of the circulating blood.

The operability of carcinoma was best determined by gastroscopy (nine cases).

Special attention is called to the fact that operation for gastric carcinoma frequently results in cure of long duration, particularly in cases in which the diagnosis is made early. Early gastroscopy together with an early roentgenogram is able to reveal operable carcinoma. Gastroscopy has proved to be superior to roentgen examination in certain cases; theoretically, in other cases, roentgen examination should be superior. The two methods are not competitors; each supplements the other, and good cooperation between the gastroscopist and the roentgenologist is essential.

Early diagnosis of gastric carcinoma can be brought about if each patient over 35 years of age who suffers from anorexia or significant loss of weight and in whom no other explanation for the symptoms is found is immediately examined gastroscopically and roentgenographically. Patients with atrophic gastritis or a benign tumor should be watched carefully by both methods. An unfavorable diffusely infiltrating carcinoma of the body of the stomach should be recognized by gastroscopy and excluded from operation. Exploratory laparotomy should be done only in those relatively few cases in which the operability of the lesion is not definitely determined by gastroscopic examination. This will result in a greatly lowered surgical mortality and will thus help to dispel the prejudice which so many hold in respect to the surgical treatment of carcinoma. This should not only bring a greater number of patients with carcinoma to the operating table but also result in the observation of a greater percentage of cases in the early stage, since a distinction will be made in the minds of the public between the prognosis of early and late cancer.

The artificial stoma was seen with the flexible gastroscope in twelve stomachs after resection and in fifteen after gastro-enterostomy; it was not seen in only three stomachs in which gastro-enterostomy had

been done, or 10 per cent of all the postoperative stomachs seen. The finding of the stoma, however, is not easy; the correct instrument must be chosen in respect to the peculiarities of the individual case.

A relatively normal stomach was seen in only three instances (10 per cent). Recurrent ulcer was found in one case and a jejunal ulcer in two cases (and probably missed in one); simple hemorrhagic erosions were observed twice. Silk sutures which had cut through the mucous membrane and were hanging free into the gastric cavity were observed three times. The most frequent disease of the postoperative stomach is chronic gastritis; it was observed in fifteen of our cases. Gastroscopic examination was unsatisfactory in four cases.

An artificial stoma (after resection or gastro-enterostomy) may remain patent or may acquire pylorus-like rhythmic activity. Four stomas of the latter type were seen: two after resection and two after gastro-enterostomy. It seems quite certain that this pylorus-like adaptation protects the stomach against the development of postoperative gastritis. Although we do not know what conditions favor this development after resection, it does seem that gastro-enterostomies which are carried out in the posterior wall near the pylorus and close to the greater curvature have a greater tendency to bring about this adaptation. Gastro-enterostomy has a further advantage in that the stoma may be closed if necessary. The activity of the gastro-enterostomy stoma in those cases in which a functional adaptation has developed is not timed in the same rhythm as is shown by the pylorus.

Silk sutures which have not been expelled into the cavity of the stomach several months after operation should be surgically removed since they irritate the mucous membrane, causing chronic gastritis with painful erosions, and may even play a part in the development of jejunal ulcers.

The observations presented in this paper do not explain the rôle played by adhesions in the development of postoperative distress.

High voltage roentgen therapy by the Coutard method causes severe gastritis in the postoperative stomach just as it does in other mucous membranes.

The illustrations in the color and figures 1, 3, 4, 5, 7, 8, 9 and 10 are from Schindler, Rudolf: Gastroscopy, Chicago, University of Chicago Press, 1937.

TRUE BRANCHIOGENIC CYST AND FISTULA OF THE NECK

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NEW YORK

In an article on "Congenital Cysts and Fistulae of the Neck," [1] published in the *Annals of Surgery* in 1932, I went into detail as to the embryologic development of the branchial apparatus and of the anlage of the thymus.

In July 1935 a boy 9 years of age was referred to me through the courtesy of Dr. Tumen, who had observed the child for a number of years. The boy was born with a small opening on the lateral side of his neck just in front of the sternocleidomastoid muscle and just below the angle of the mandible, on a level with the hyoid bone. It was first noticed when he was 5 months old, when it was an opening the size of a pinpoint. The mother stated that colorless matter came out. A swelling developed below the left ear, and when the boy was 18 months old the swelling was lanced by a physician. It subsided, and the wound closed. After this at intervals the swelling would recur and open by itself, and a discharge would come through the previous incision. Three or four times in the next few years it was necessary to lance the swelling. When the boy was 4 years old it recurred worse than before, and he was operated on by another surgeon, who tried to dissect out the tract. Apparently, from the description as given by the parents and Dr. Tumen, at this operation an opening was made into the trachea, and the operating surgeon believed that the tract was connected with the trachea. The wound finally healed, but the same sequence of events continued for the next five years, until the present time. Dr. Tumen injected dye into the fistulous opening, and, as shown by the accompanying roentgenograms, it went upward in the direction of the tonsillar fossa (fig. 1). In view of this picture and the history and the studies of Wenglowski, as published in my article in 1932, I made a preoperative diagnosis of probable lateral cyst and fistula of the neck, a remnant of the pharyngothymic duct, and I felt that on operation I would probably find the tract running upward toward the tonsillar fossa and entering the pharynx through the bed of the removed tonsil or close to the posterior pillar. From the studies of the embryology of the branchial apparatus and the pharyngothymic duct, I definitely felt that there was no connection between the tract and the trachea.

In July 1935 the boy was operated on, tribromethanol in amylene hydrate supplemented by a general anesthetic being used. The findings at the time of operation were exceedingly interesting. The fistulous opening was circumscribed, and the

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The case reported in this paper was presented before the New York Surgical Society, April 22, 1936.

1. Meyer, Herbert Willy: Congenital Cysts and Fistulae of the Neck, Ann. Surg. **95**:226-248 (Feb.) 1932.

infiltrated tissue surrounding the fistulous tract was dissected free from the sternocleidomastoid and the platysma myoides muscle. As expected, it did not run toward the trachea. There was an infiltrated area, that passed upward and somewhat posteriorly toward the ear, but this could easily be dissected free, and it was found that the main portion of the tract, which was thick and dense, passed upward and somewhat inward into the jugular fossa, but then, to my surprise, instead of curving forward toward the pharynx and tonsillar fossa it curved upward and backward to the very apex of the jugular fossa. The tract was firmly and densely adherent to the tip of the styloid process. As a matter of fact, it was a part of the styloid process, and in order to effect complete removal of the tract it was necessary to bite off the tip of the styloid process (fig. 2 *A*). This permitted complete and radical removal of the entire congenital tract. With closure of the wound an uneventful recovery took place, and two years later there had been no recurrence of the previous trouble.

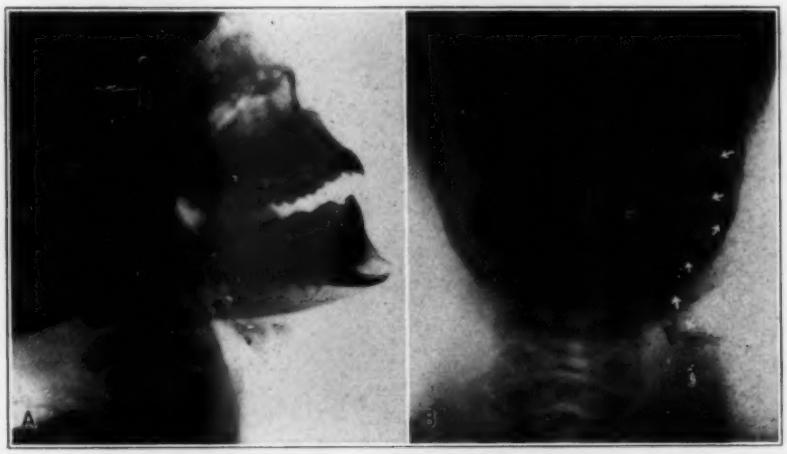

Fig. 1.—*A*, lateral roentgenogram showing the path taken by dye which was injected into the fistula. *B*, anteroposterior view. A tract is seen running upward toward the tonsillar fossa.

EMBRYOLOGY

A review of the embryology of this region was then made, and the facts as laid down by Wenglowski and as published by me in 1932 were again studied.

In an embryo of 6.5 mm. the first branchial arch goes to make up the upper jaw and the anterior portion of the lower jaw. The second arch goes to make up the operculum. The third and fourth arches do not meet in the middle. The first branchial cleft goes to form the external auditory canal. The second, third and fourth clefts are also visible. All of these branchial arches and clefts are covered with squamous epithelium.

The pharyngeal arches correspond to the branchial clefts. The first goes to make up the body of the tongue. The second, third and fourth

arches are visible. The pharyngeal pouches correspond to the branchial arches. The first goes to make up the middle ear and the lobe of the ear, and the third, the anlage of the thymus.

In an embryo of 8 mm. the first branchial arch goes to make up the lips and the jaws. The second, third, fourth, fifth and sixth arches are visible. The branchial clefts are narrowed by the growth of the arches. The first cleft goes to make up the external auditory canal. Here the sinus cervicalis is deep and large.

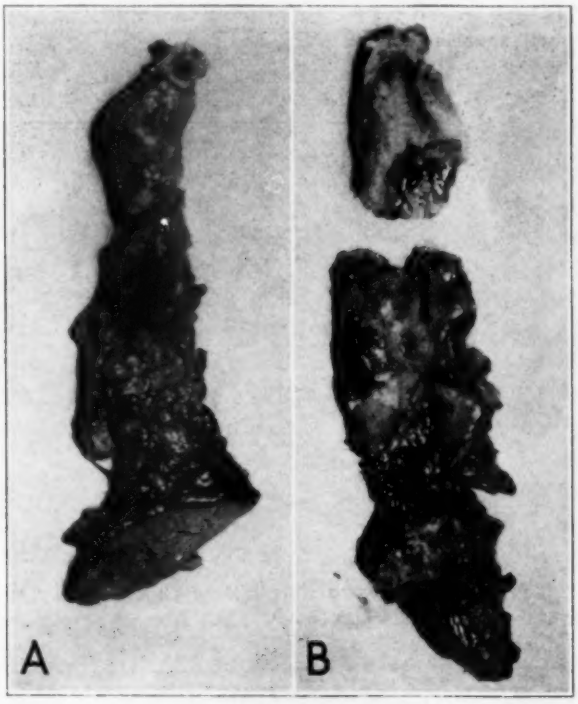

Fig. 2.—*A*, specimen removed at the time of radical excision, showing skin at the lower portion of the picture at the site of the external opening. A portion of the tip of the styloid process is at the uppermost portion of the photograph. *B*, the cyst laid open. Note the removal of skin with fine hairs in the upper section of the tract close to the styloid process.

The pharyngeal arches, one to four, go to make up the tongue. The pharyngeal pouches show the third to be the anlage of the thymus, while the fourth is the anlage of the lateral lobes of the thyroid. These pouches are covered by ciliated epithelium.

In an embryo of 11 mm. retrogression of the branchial apparatus takes place. The sinus cervicalis is closed from without, and only three arches are visible. The first branchial arch goes to make up the lower lip and the anlage of the jaw. The second branchial arch, which is

conical, meets the opposite one in a narrow strand. The third branchial arch is not large and joins with the neck, displacing the entrance into the sinus cervicalis. The branchial clefts are becoming flat and narrow.

In embryos of 12 and 13 mm. further retrogression takes place. The first and second branchial arches are visible. The medial ends join with the neck, and the contour of the neck becomes visible.

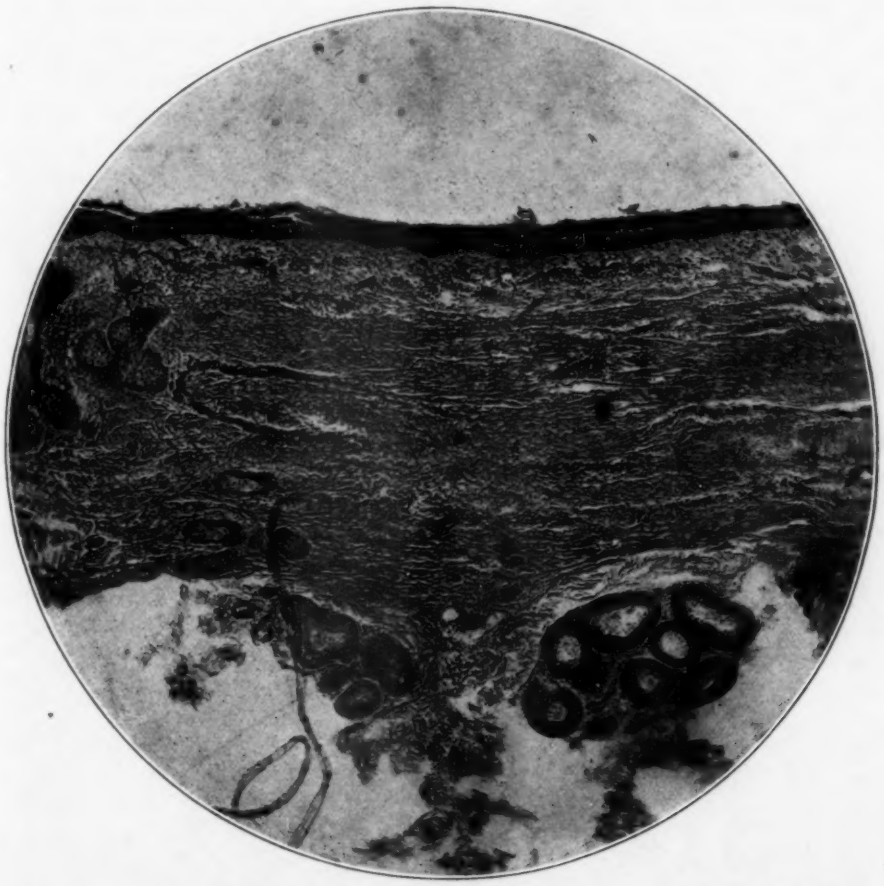

Fig. 3.—Section through the inner lining of the cyst close to the styloid process, showing sweat glands and normal epithelium.

COMMENT

The beginning of the branchial apparatus takes place in the second half of the first month of embryonic life. In the course of the second month the branchial apparatus completely disappears. In other words, it lasts only one month. According to Wenglowski, each branchial arch and cleft crosses the pharyngeal arch and pouch. His stated that the

branchial arch coincides with the pharyngeal arch and the branchial cleft with the pharyngeal cleft. Wenglowski definitely disagreed with this.

The branchial apparatus runs forward, upward and orally, while the pharyngeal apparatus runs posterior, downward and aborally. The arches grow closer and closer, until the clefts disappear and the epithe-

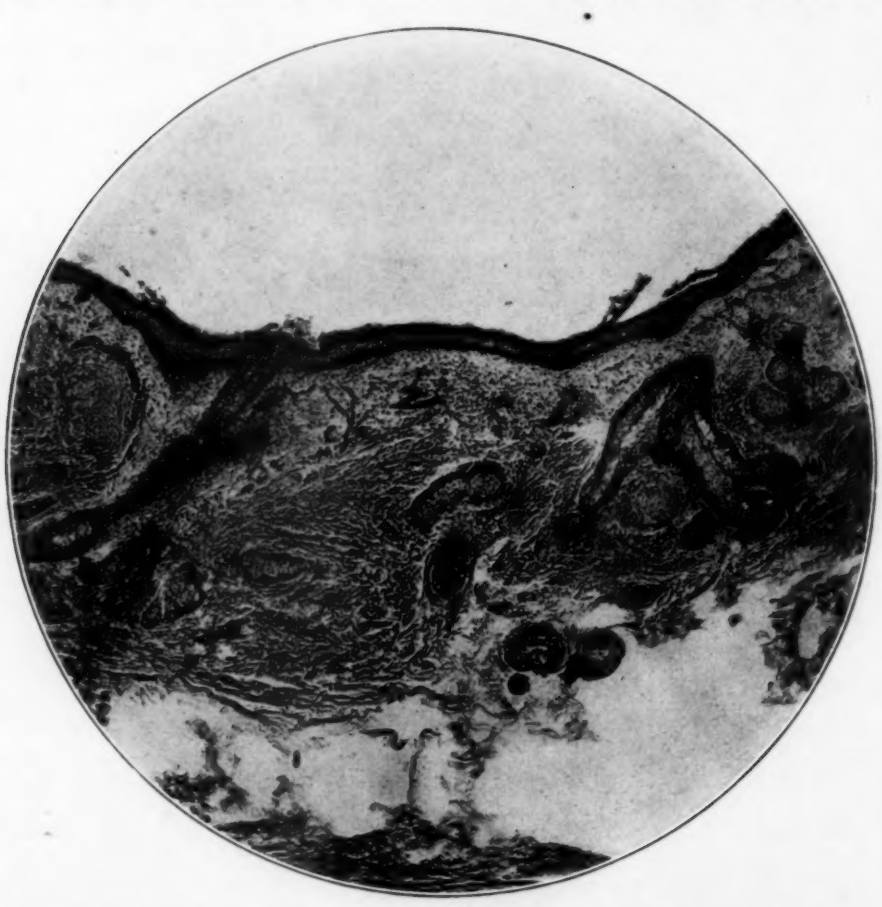

Fig. 4.—Section through the inner lining of the cyst close to the styloid process, showing hair follicles and sebaceous glands in the wall of the cyst.

lium is displaced outward by the mesenchymal growth or by adhesion and obliteration.

In development the first arch goes to form the lateral portion of the upper lip and jaw, the lower lip and jaw and the body of the tongue. The second arch goes to make up the body of the hyoid, the stylohyoid ligament and muscle and the anterior portion of the base of the tongue and the arcus palatoglossus. The third arch goes to form the greater

cornu of the hyoid bone, the posterior portion of the base of the tongue and the arcus palatopharyngeus. The fourth, fifth and sixth arches go to make up the soft parts of the neck surrounding the greater cornu of the hyoid bone.

The first cleft goes to make up the external auditory canal and the lobe of the ear; the second cleft, the tonsillar fossa; the third cleft, the thymus, and the fourth cleft, the lateral lobes of the thyroid.

The branchial apparatus belongs to the head and not to the neck. Any congenital pathologic condition referable to the branchial apparatus in human beings must rest along the lower jaw and around the hyoid bone and the cornu of this bone. Nothing below the lower level of the hyoid bone has any genetic relation with the branchial apparatus. All congenital anomalies caused by incomplete retrogression of the branchial apparatus must be located in the region around or above the lower border of the hyoid bone. Any congenital anomaly below this level is in definite relationship with the pharyngothymic duct and must be classified as a lateral cyst or fistula originating from the pharyngothymic duct.

In the case reported here, after the removal of the specimen the tract was opened and found to be lined with normal-looking skin and to be firmly attached to the styloid process (fig. *2 B*). Microscopically, as shown in the photomicrographs, the wall of this tract contained all the appendages of the skin as hair follicles, sweat glands and sebaceous glands (figs. 3 and 4). In other words, the growth was a true branchiogenetic cyst and tract originating from the second branchial arch. Complete removal of this, of course, brought about a cure of the condition. It seems of interest to publish once more in résumé the embryology of this region, which is always interesting and fascinating. Any reader who is interested in a greater detail of these conditions is referred to the previous article by me which explains the aforementioned facts.[1]

LAXITY OF THE RADIO-ULNAR JOINT FOLLOWING COLLES' FRACTURE

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Since Sir Astley Cooper's [1] first description of the lesion in 1842, the literature has been replete with reports of frank dislocations at the lower radio-ulnar articulation associated with Colles' fracture. The more frequent but less severe degrees of relaxation in that joint, however, have received scant attention. In approximately one of every nine cases, Colles' fracture is followed after the healing period by laxity of the radio-ulnar joint. In some of these instances slight radio-ulnar diastasis is present, causing broadening of the wrist and apparent radial deviation of the hand. In others, the bones remain loosely in their normal position, and no deformity is apparent. In some cases the condition is entirely symptomless. In many, however, its association with lasting weakness, limitation of rotation and pain in the ulnar aspect of the wrist joint is of sufficient regularity to indicate a causal relationship which merits investigation.

Many observers have noted the persistence of these phenomena after Colles' fracture, but there has been little agreement about their interpretation or importance. In 1915 Darrach and Dwight [2] called attention to the severity and frequency of symptoms referable to the radio-ulnar joint following Colles' fracture, which they believed resulted from injury to the radio-ulnar ligaments. They maintained that with perfect reduction the normal relationship was regained and injury to the joint structures repaired. Additional support to the radio-ulnar joint, such as a leather wristlet, was advised after splinting, and in the event of persistent symptoms, subperiosteal resection of the head of the ulna was recommended. In 1917 Pilcher [3] wrote: "The projection of the end of the ulna on the inner anterior aspect of the wrist is the most noticeable and frequent permanent sequel of this fracture." Later, he added: "The [ulnar] projection may persist even when there is no appreciable deformity of the radius, through lack of full restoration of the close ligamentous bands that before the injury tied it to the radius." Pilcher,

1. Cooper, Astley: A Treatise on Dislocations and on Fractures of the Joints, ed. 2, London, J. Churchill, 1842, pp. 490, 496 and 497.

2. Darrach, W., and Dwight, K.: Derangements of the Inferior Radio-Ulnar Articulation, M. Rec. **87**:708 (April 24) 1915.

3. Pilcher, L. S.: Fractures of the Lower Extremity or Base of the Radius, Ann. Surg. **65**:1, 1917.

however, was not impressed by the "slight if any" symptoms, which follow this malposition. In 1921 Hitzrot and Murray [4] reviewed one hundred and ninety-six cases of Colles' fracture, noting the frequency of residual symptoms referable to the radio-ulnar joint. They did not attempt an explanation of these symptoms but reached the conclusion that functional disturbance is more marked in the more severe injuries and that early and complete anatomic restoration gives better functional results. On the basis of a review of the late results in four hundred and twenty-four cases of Colles' fracture, Edwards and Clayton [5] found that from two to four years after injury 23.1 per cent of the patients complained of some residual symptoms, among which pain or weakness on playing tennis or after heavy manual labor and tenderness over the internal lateral ligament were common. The symptoms were attributed to involvement of the radio-ulnar joint in the fracture, although roentgenographic evidence did not confirm this correlation. Grasby and Trick,[6] in a significant review of fifty cases in which fracture had been sustained from two to four years previously, found that when displacement was a marked feature the end-results were invariably unsatisfactory, owing to symptoms referable to the radio-ulnar joint. These workers concluded that the symptoms were due to arthritis at that joint secondary to incomplete reduction of the fracture. Bankart [7] agreed with their interpretation. The report of the Fracture Committee of the American College of Surgeons [8] mentioned the frequent prominence of the head of the ulna and pain referred to the region of the styloid process after healing of even a well reduced fracture. No suggestions regarding etiology or therapy were made. Moorhead [9] stated: "There is an inter-articular fibro-cartilage at the lower end of the radius, and if this is involved in the fracture, we are almost certain to have an eversion deformity despite anatomical setting of the bone itself." Ghormley [10] mentioned pain referable to the styloid process of the ulna as a troublesome complication following Colles' fracture but expressed the belief that it can be remedied by removal of the fractured styloid process of

4. Hitzrot, J. M., and Murray, C. R.: The Factors That Influence Prognosis in Fractures at the Base of the Radius, Am. J. Surg. **35**:17, 1921.

5. Edwards, H., and Clayton, E. B.: Fractures of the Lower End of the Radius in Adults, Brit. M. J. **1**:61 (Jan. 12) 1929.

6. Grasby, E. D., and Trick, S. R.: Investigation of the End-Results of Colles' Fractures, Brit. M. J. **1**:391 (March 2) 1929.

7. Bankart, A. S. B.: Treatment of Colles' Fracture, Brit. M. J. **1**:491 (March 16) 1929.

8. American College of Surgeons Fracture Committee: Principles and Outline of Fracture Treatment, Bull. Am. Coll. Surgeons **15**:3, 1931.

9. Moorhead, J. J.: Traumatotherapy: The Treatment of the Injured, Philadelphia, W. B. Saunders Company, 1931.

10. Ghormley, R. K., and Mroz, R. J.: Fractures of the Wrist, Surg., Gynec. & Obst. **55**:377, 1932.

the ulna. Magnuson [11] considered the throwing out of alinement of the radio-ulnar joint and the tear or overstretching of its ligaments a serious source of disability unless "reduction is accomplished." Platt,[12] in a recent consideration of these disturbances of the inferior radio-ulnar articulation after Colles' fracture, wrote as follows:

> In not less than fifty per cent of Colles' fractures, the fracture line on its mesial aspect reaches the upper limit of the articular facet for the head of the ulna, or enters the joint lower down. I have not been able to satisfy myself that involvement of the radio-ulnar joint in this fashion materially affects the function of rotation of the forearm, provided the injured joint is mobilized with discretion. Much more serious, however, is the disturbance which may result from a fracture of the ulnar styloid process at its base, or the equivalent injury—detachment of the fibro-cartilage from the lower end of the ulna. As regards the frequency of this latter complication, I have no statistical data, for clinical proof of its existence is rarely forthcoming. This point in the anatomy of Colles' fracture has exercised the minds of surgeons for many generations.

Scudder,[13] Key and Conwell,[14] Preston [15] and a few others called attention to widening of the distal radio-ulnar joint after Colles' fracture and suggested that symptoms referable to the joint are liable to ensue unless the displacement is reduced. In many other comprehensive texts and reviews no mention is made of the complication or its sequelae.

This brief survey of the recent literature suffices to point out that in the experience of many observers symptoms referable to the radio-ulnar joint and deviations of the ulna are not uncommon sequelae of Colles' fracture, particularly when displacement has been severe and reduction incomplete. That some have laid less emphasis on these complications is readily understandable when their divergent sources of material are considered—a divergence sufficient to account for variation of from less than 10 per cent to more than 70 per cent in the occurrence of coincident avulsion of the styloid process of the ulna.

In these studies the symptoms have been attributed to a wide range of disturbances of the radio-ulnar joint; to malalinement of the joint surfaces, to direct involvement of the joint in the fracture line, to arthritis, to rupture of the articular disk, to fracture of the styloid process of the ulna, to sprain of the ligaments of the radio-ulnar joint and, vaguely, to residual malposition.

On careful scrutiny, many of these hypotheses are untenable. Persistent symptoms referable to the radio-ulnar joint and pain on the

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11. Magnuson, Paul B.: Fractures, Philadelphia, J. B. Lippincott Company, 1933.

12. Platt, H.: Colles' Fracture, Surg., Gynec. & Obst. **60**:542, 1935.

13. Scudder, C. L.: Treatment of Fractures, Philadelphia, W. B. Saunders Company, 1931.

14. Key, J. A., and Conwell, H. E.: The Management of Fractures, Dislocations, and Sprains, St. Louis, C. V. Mosby Company, 1934.

15. Preston, M. E.: Fractures and Dislocations, St. Louis, C. V. Mosby Company, 1915.

ulnar aspect of the wrist have been observed with and without fracture of the styloid process of the ulna and often, in the complete absence of stiffness of the finger and wrist joints or other arthritic manifestations. Roentgenograms have shown that direct involvement of the articular surface of the radio-ulnar joint in the fracture line can account for but a small proportion of these cases (Hitzrot and Murray and Edwards and Clayton). The greater frequency with which pain in the radio-ulnar joint occurs after incomplete reduction of a fracture has led some to infer that the pain was in some way directly referable to the residual displacement and could have been avoided by more perfect reduction. It is impossible to reconcile their conclusions with the fact that symptoms referable to the radio-ulnar joint, though less frequent, are often observed after faultless reposition and are absent in many striking cases of malposition.

It appears more reasonable, therefore, to regard the symptoms as due to a lesion of the distal radio-ulnar structures caused by displacement of the base of the radius at the time of injury, a lesion which sometimes, though not always, heals after perfect reposition of the bony fragment. The less frequent occurrence of symptoms referable to the radio-ulnar joint in cases of well reduced fracture is accounted for by the inclusion in this number of many cases in which the original bony displacement was insufficient to cause injury to the radio-ulnar ligaments as well as by occasional healing of these structures when the bone is well reduced.

In further support of this contention is the fact that in my own experience there is only one objective finding which has almost invariably characterized this condition, i. e., loss of stability at the distal radio-ulnar joint. In my opinion, this association and the foregoing findings show clearly the essential dependence of the symptoms on damage to the radio-ulnar ligaments sustained by the original trauma and indicate that even perfect reduction of the fracture cannot by itself be relied on to restore the stability of the radio-ulnar joint completely once the ligaments of this joint have been severely damaged.

The following studies were undertaken with the hope of clarifying the concept of the mechanism, prophylaxis and therapy of this condition.

ANATOMY

The articular surfaces composing the inferior radio-ulnar articulation are maintained in normal relationship by three ligamentous structures, the dorsal and ventral radio-ulnar ligaments and the triangular fibrocartilage or articular disk. The dorsal ligament is a weak structure reenforcing the dorsal joint capsule. It stretches from the posterior margin of the articular notch of the radius to the posterior surface of the head of the ulna. The ventral ligament is a similar mechanism

extending between corresponding surfaces on the ventral aspect. The articular disk is a triangular band of fibrocartilage, the base of which is attached to the lower margin of the articular notch of the radius. Its apex attaches to a short ligament, which in turn inserts into the base and the inner surface of the styloid process of the ulna and the internal lateral ligament (fig. 1).

This mechanism is so adjusted that in most persons motion is entirely limited to the gliding of the sigmoid cavity of the radius about the articular surface of the head of the ulna. Occasionally, a slight and symptomless degree of laxity is present. Stability of the distal radio-ulnar joint is maintained in all rotary positions of the forearm by variation in the tension of these ligaments. The dorsal radio-ulnar ligament is spanned in pronation and relaxed in supination. The ventral

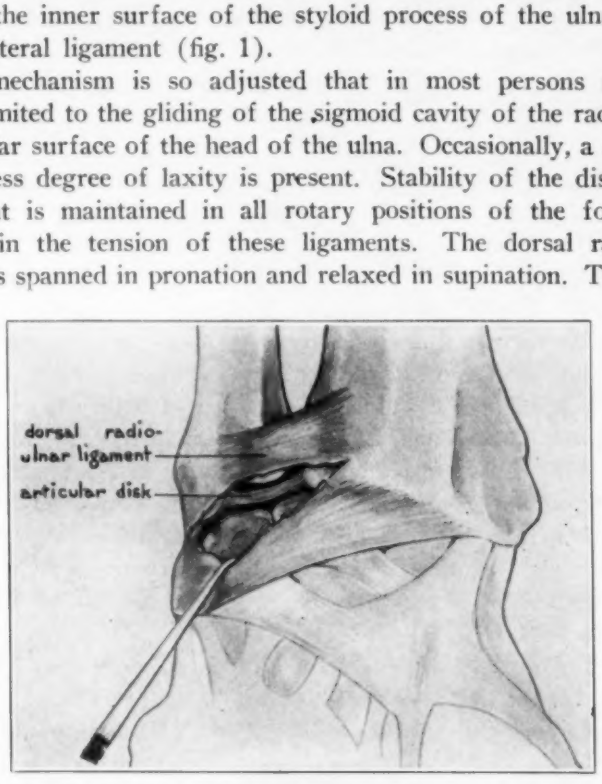

Fig. 1.—Diagram showing the radio-ulnar ligament and articular disk.

radio-ulnar ligament is relaxed in pronation and tensed in supination. The articular disk is fairly taut in all rotary positions, its anterior fibers tightening in supination and its posterior fibers, in pronation. In pronation, then, the stability of the joint is maintained by the dorsal radio-ulnar ligament and the articular disk, and since Colles' fracture is practically always sustained with the forearm prone, these ligaments which are tensed in that position are the ones jeopardized by displacement of the radial fragment (fig. 2).

Anatomically, the articular disk is more vulnerable than the dorsal radio-ulnar ligament. The broad, firm fixation of its base to the base of the radius prevents any considerable degree of forward and backward sliding. It cannot be displaced upward because the head of the ulna is directly above. Moreover, it is located where the greatest radio-ulnar diastasis exists when the radial fragment is angulated and displaced upward. For these reasons, anything more than minimal upward

and backward displacement of this fragment cannot occur without producing at least a partial tear of this structure (or its equivalent, avulsion of the styloid process of the ulna).

The dorsal radio-ulnar ligament appears to be better protected. If the arm is not in pronation at the time of fracture, this ligament is relaxed and so may escape injury. The height of its upper radial attachment is subject to considerable normal variation but is seldom less than ¾ inch (1.9 cm.) above the highest point of the wrist joint. This level is higher than that at which Colles' fracture occurs in most instances, and for this reason a good portion of the ligament may remain attached to the shaft and so be preserved from injury when the distal fragment is displaced. The structure is not strong, and rupture of its fibers is anatomically possible, even in a low fracture, if the ligament is tightly spanned by pronation of the forearm (fig. 3).

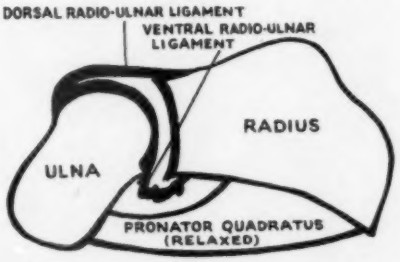

Fig. 2.—Diagrammatic cross-section of the distal radio-ulnar joint in pronation, showing tension of the dorsal radio-ulnar ligament and relaxation of the ventral radio-ulnar ligament.

EXPERIMENTS ON CADAVERS

With these facts in mind, experiments on cadavers were undertaken to determine the relative importance of the articular disk and the dorsal radio-ulnar ligament in the maintenance of fixation of the distal radio-ulnar joint.

(*a*) In the forearm of a cadaver the insertion of the articular disk into the styloid process of the ulna was severed with a tenotome. A minimal degree of laxity of the radio-ulnar joint in the prone position and a slightly greater degree in the midposition followed this operation. The lower half of the dorsal radio-ulnar ligament was then cut with the forearm in pronation. The lower end of the ulna could now be freely moved up and down with reference to the rest of the forearm. Complete sections of the dorsal radio-ulnar ligament permitted a still greater range of motion, and with the arm in pronation it was now possible to dislocate the lower end of the ulna completely. In supination the tension of the ventral ligament returned the ulna to its normal

position and held it firmly there. This experiment was repeated in two other forearms with identical results.

(*b*) In each of three forearms the dorsal radio-ulnar ligament was sectioned and the articular disk permitted to remain intact. In all cases this produced moderate laxity at the radio-ulnar joint in the prone position. When the forearm was in full supination, the edges of the tear in the dorsal radio-ulnar ligament were approximated, and complete stability was restored.

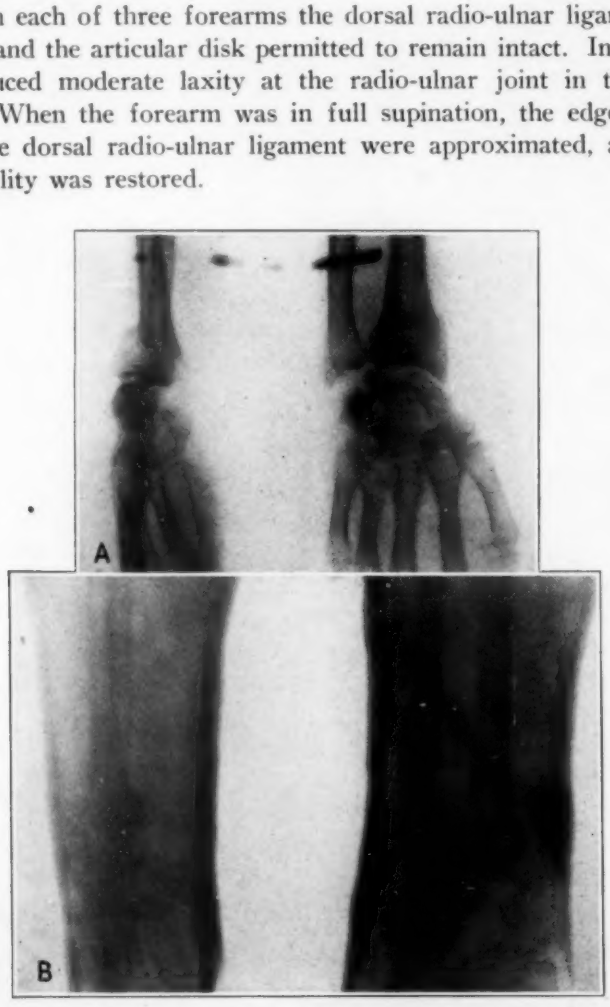

Fig. 3 (M. B.).—*A*, roentgenogram illustrating a high type of Colles' fracture which jeopardizes radio-ulnar ligaments. The radio-ulnar joint is not involved in the fracture line. *B*, roentgenogram showing perfect bony reduction, in spite of which symptoms referable to the radio-ulnar joint have persisted for over one year.

These findings may be summarized as follows:

1. With the forearm prone, section of the articular disk alone results in minimal laxity of the radio-ulnar joint.

2. With the forearm prone, section of the dorsal radio-ulnar ligament results in moderate laxity. (As the articular disk is more vul-

nerable than the dorsal radio-ulnar ligament, this condition must be clinically rare.)

3. Section of the articular disk plus partial section of the dorsal radio-ulnar ligament increases the degree of laxity of the radio-ulnar joint.

4. Complete section (of both the articular disk and the radio-ulnar ligament) permits great freedom of motion between the distal end of the radius and the ulna, so that in pronation complete dorsal dislocation of the head of the ulna is possible.

5. The position of supination reduces any displacement of the ulna and approximates the torn edges of the dorsal radio-ulnar ligament.

These findings justify certain clinical inferences which clarify the concept of the lesion. Primarily, since the degree of laxity of the radio-ulnar joint after Colles' fracture is considerably greater in most cases than that elicited by section of the articular disk alone, it may be safely assumed that in these cases the integrity of the dorsal radio-ulnar ligament is also compromised. Complete avulsion of the dorsal radio-ulnar ligament is not necessary to produce a moderate degree of laxity, and it is likely that in most cases a partial tear or sprain in this ligament plus a partial or complete avulsion of the articular disk or the styloid process of the ulna constitutes the pathologic basis for laxity after Colles' fractures.

On this basis may be explained the absence of perceptible laxity of the radio-ulnar joint in many cases of Colles' fracture in which the styloid process of the ulna is avulsed at the base and consequent complete loss of function of the articular disk must occur. Although the styloid process of the ulna is fractured in well over half of all cases of Colles' fracture, preservation of the dorsal radio-ulnar ligament suffices to retain stability of the radio-ulnar joint. Dissections on cadavers have indicated that the dorsal radio-ulnar ligament may be preserved if the fracture is low or if displacement is minimal, and, clinically, fractures with these characteristics are seldom complicated by laxity or its attendant symptoms.

Of greater practical importance is the freedom of motion permitted by the intact ventral radio-ulnar ligament in the prone position after the dorsal radio-ulnar ligament and articular disk are sectioned. This laxity of the ventral ligament in pronation accounts for its preservation in all but the most extensive Colles' fracture-displacements, a circumstance which permits its utilization in practically all cases to restore the integrity of the radio-ulnar joint. The supine position, by spanning this ligament, draws the head of the radius and of the ulna together. In this position the pronator quadratus muscle is also stretched, so that it can actively assist in this function. Moreover, supination relaxes both the dorsal radio-ulnar ligament and the posterior portion of the

articular disk, so that any torn edges may be approximated, and healing may occur without distortion. These observations clearly indicate that from an anatomic standpoint supination is the position of choice for the treatment of injury of the radio-ulnar ligament accompanying Colles' fracture.

SYMPTOMS

Laxity of the radio-ulnar joint may be entirely asymptomatic or may be the cause of severe disability. When pain is present, it ranges from a dull ache on the ulnar aspect of the wrist accompanying and following function to recurrent acute paralyzing twinges. Pain of the latter character is particularly associated with pronation against resistance, and its severity is sometimes such that any object in the hand is immediately dropped. Usually these pains are most severe during the first few months of convalescence, after which their intensity diminishes to a less severe but constant level. On the other hand, pain sometimes increases with the resumption of full activity, so that it becomes most troublesome four or five months after the time of fracture.

On examination the wrist joint may appear slightly broadened owing to mesial displacement of the head of the ulna. In other instances the ulna remains loosely in its normal position, so that no deformity is apparent. Tenderness over the dorsal radio-ulnar ligament, below and on the lateral aspect of the wrist joint, is usually present. Limitation of flexion and extension at the wrist and fingers is not referable to laxity of the radio-ulnar joint. Supination is usually limited, but both active and passive pronation cause severe pain at its limit. In cases of recent fracture all of these objective findings are obscured by the proximity of the fracture hematoma and reaction. Slight radio-ulnar diastasis may or may not be visualized by roentgenographic examination, and it must be emphasized that films cannot be relied on for diagnosis of this condition.

Immediately after reduction of the bony displacement or at any time thereafter the condition may be recognized with ease if the radio-ulnar joint is examined for stability, but only by this procedure. One hand of the examiner fixes the radius and hand of the patient firmly in pronation so that no rotation is possible. With the thumb and forefinger of the other hand, the head of the ulna is gripped, and an effort is made to displace it upward and downward. The degree of free motion so elicited may be compared with that in the opposite wrist as a control.

The disability caused by laxity of the radio-ulnar joint is exceedingly difficult to evaluate. No definite standards of efficiency can be employed when the demands of the patient's occupation on the function of the wrist are at such wide variance. Moreover, a lesion of the right wrist generally causes much greater disability than a similar left-sided lesion, a fact which is not accorded due consideration in the evaluation

of the results of Colles' fracture. As most of the patients with this type of fracture are able to resume their former occupations more or less well, the disability in most instances is considered as relatively minor. However, within the past year, the following typical complaints were received. One carpenter was able to perform his usual functions fairly well, except for complete inability to use a screw-driver because of pain. Two tennis players were compelled to abandon the game because of severe pain at every powerful stroke. A middle-aged woman complained of severe pain on the ulnar aspect of the joint, weakness and swelling after sewing or knitting for a short time. Two other women suffered similarly after a few hours of household duties. These illustrative complaints were chosen from patients whose injuries had been sustained at least eight months previously. While other findings in these cases were variable, the radio-ulnar joint in all instances was unstable and, with the wrist prone, the head of the ulna could be freely moved with reference to the base of the radius.

PROPHYLAXIS

Complete reduction of a fracture is of maximal importance for restoring the normal relationship of the distal radio-ulnar joint and constitutes the first step in the prevention of permanent laxity of the joint. Evidence from all sources indicates the greater frequency with which symptoms follow incomplete reductions. The greater severity of symptoms in the incompletely reduced fractures is illustrated in the appended table.

Directly after the accomplishment of reduction, the radio-ulnar joint should be tested for stability. If stability is present at this examination, subsequent laxity need not be feared, and the customary forearm splint suffices for immobilization. If instability is discovered, the forearm should be rotated into full supination and maintained in that position by inclusion of the upper part of the arm in the immobilizing device for a period of from three to four weeks. The position of supination, by tensing the ventral radio-ulnar ligament and spanning the pronator quadratus muscle, draws the muscles of the radius and ulna together. It relaxes the dorsal radio-ulnar ligament and posterior fibers of the articular disk, so that torn edges may be approximated during the process of healing.

If the arm is not fixed in supination but is permitted to rotate after reduction, it has a tendency to fall into pronation. The dorsal radio-ulnar ligament is thus subjected to continuous or intermittent tension, and healing with elongation must be anticipated.

It is interesting that with the wrist in supination the displacement of an avulsed styloid process of the ulna is reduced (fig. 4), and if

Data on Symptoms Referable to the Radio-Ulnar Joint After Colles' Fracture

				Original Fracture Data					Present Status				
										Objective Findings			
Patient	Age	Sex	Arm	Displacement	Level	Styloid Process of Ulna	Reduction	Duration	Persistent Symptoms	Arthritis	Broadening of Wrist	Pain on Pronation	Radio-Ulnar Laxity
H. S.	24	F	L	?	High	—	Excellent	6½ yr.	Wrist weak since fracture; pain became severe when nursing commenced one year ago; since then constant pain during working hours on ulnar aspect and over radio-ulnar joint	.....	Slight	Slight	+
L. R.	28	M	R	Marked	High	+	Good	3¼ yr.	Patient unable to play tennis since onset because of acute sharp twinges of pain; occasional pains at other times but not severe; persistent weakness	.....	.....	Very slight	+
M. L.	71	F	R	?	Medium	—	Poor	3 yr.	Arthritic pains and limitation of wrist and finger motion severe for 4 months after fracture; since then, localization at radio-ulnar joint and ulnar aspect; unable to sew or knit	+ Fingers and wrist	Marked	Severe	+
B. T.	47	F	R	Moderate	High	+	Fair; ⅛ inch (0.3 cm.) radial shortening	2½ yr.	Result satisfactory but for weakness and pain after working (housework); condition has persisted without change for 2 years	.....	Slight	Severe	+
B. R.	52	F	L	Marked	High	+	Poor; ¼ inch (0.6 cm.) radial shortening	2 yr.	Very severe pain over radio-ulnar joint and ulnar aspect of wrist—constant and with acute twinges unless complete rest is observed; unable to sew for 5 minutes	+ Slight; wrist	Marked	Severe	++
C. B.	35	M	R	Marked	High	—	Excellent	2 yr.	Slight weakness; repeated acute twinges of paralyzing pain of increasing frequency at any motion of twisting against resistance	.....	.....	Very slight	+
B. P.	54	M	R	Marked	High	—	Good	1¼ yr.	Working as carpenter but has pain when he has to use tools requiring a twisting motion; pain is chiefly on ulnar aspect of wrist	.....	Slight	Moderate	+
M. S.	31	M	R	Marked	Medium	+	Fair; ⅛ inch radial shortening	1 yr.	Persistent weakness; sudden acute pains at radio-ulnar joint on athletic activity	.....	.....	.....	+
N. C.	56	F	R	Moderate	High	+	Poor; ⅛ inch radial shortening	9 mo.	Pains of moderate severity on working and for several hours thereafter; no acute twinges; disability not great; pain most severe over radio-ulnar joint	.....	Slight	Severe	+

sufficient time is given, it may reunite with the shaft so that complete restitutio ad integrum of the joint is established. Practically, it has not been deemed advisable to maintain supination long enough to permit such bony union to occur.

In certain instances Colles' fracture cannot be completely reduced owing to telescoping of the bony structure of the head of the radius. As in cases in which the fracture is well reduced, when the fracture is unassociated with laxity of the radio-ulnar joint its late occurrence need not be feared, and the employment of the supinated position is unnecessary. Those fractures which, for this or other reasons, have not been well reduced and manifest laxity of the radio-ulnar joint constitute the greatest source of late symptoms referable to this joint.

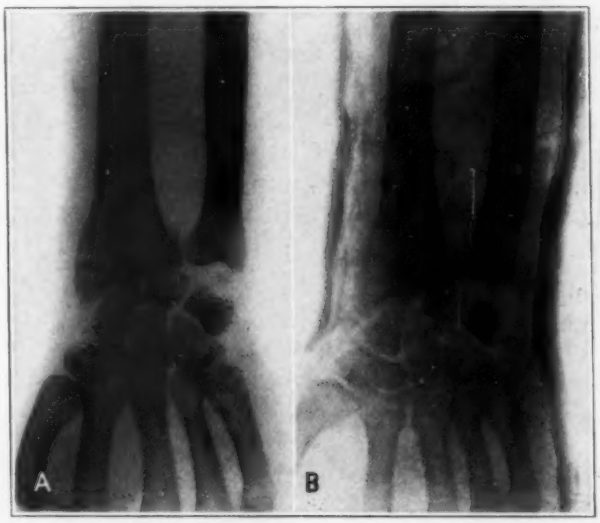

Fig. 4.—*A*, a roentgenogram showing reduction of Colles' fracture in pronation, and *B*, one showing reduction of the displaced styloid process of the ulna after immobilization in supination.

Upward displacement of the base of the radius in such fractures entails equivalent displacement of the fragment of dorsal radio-ulnar ligament attached. The resulting separation of the ligament's torn edges precludes healing, a fact which probably accounts for the frequency of late symptoms referable to the radio-ulnar joint. Approximation of the torn structure can be attained only by assumption of the supinated position.

Two theoretical objections to the employment of supination in the treatment of Colles' fracture merit consideration. The desirability of limiting rotational movement for several weeks in persons with arthritis is questionable. It is my opinion that in such cases the relative advantages of permanent relaxation of the radio-ulnar joint as opposed to

prolonged stiffness of the joint must be individually evaluated. It has been suggested that the reduction of the fracture attained with the arm in pronation might be jeopardized by subsequent rotation into supination. To determine whether such slipping might occur, ten fractures were checked by roentgenograms after reduction and again after supination. In no instance had supination affected the position of the bony fragment.

Although the recovery of full active rotation is somewhat delayed by fixation in supination, stability of the radio-ulnar joint is present when the splints are removed, and the danger of permanent pain or deformity from this source has been avoided.

In the past two years the foregoing technic has been employed on twelve cases of Colles' fracture in which, after reduction, there was laxity of the radio-ulnar joint. Immediately on removal of the fixation from the upper part of the arm, five of the patients were able to rotate the hand freely and without pain. In the remainder, the return of free rotation was delayed from one to three weeks. Except in one case (that of a severely comminuted fracture in which it seemed that rupture of both ventral and dorsal ligaments had occurred) stability was present when the plaster was removed. In these eleven cases the joints have remained stable and painless. Although a detailed report on the end-results must be deferred for several years, at the present time it can be definitely stated that in these patients instability of the radio-ulnar joint has been prevented, prominence of the head of the ulna has been greatly minimized and freedom from symptoms referable to the radio-ulnar joint has been complete.

THERAPY

The treatment of laxity of the radio-ulnar joint is determined by the duration, the course and the severity of the symptoms. When the wrist is painful only during and after certain activities, a 2 inch (5 cm.) broad leather wristlet worn during these times can give considerable relief. That this device is not more effective may be attributable to the fact that the forearm measures slightly less in circumference when it is in pronation. As a consequence, the wristlet is more slack in that position in which its support is most necessary. An elastic wristlet has not sufficient strength to be of value.

Radical treatment is indicated when symptoms have continued for at least four months after injury and when, in spite of judicious conservative treatment, they have exhibited no tendency to subside. The following procedure has, in my experience, proved adequate when there is no gross bony distortion of either articular facet

A dorsolateral incision 1½ inches (3.8 cm.) in length is made at the level of the radio-ulnar joint, and the dorsal radio-ulnar ligament

is exposed. The ligament is usually found to be redundant and scarified. All scar tissue is excised, and, through this approach, the radio-ulnar joint surfaces are exposed. Any granulation tissue between the head of the ulna and the radius is then curetted out. If the triangular fibrocartilage is distorted or fractured, this structure or any part of it may be freely removed. It may be recalled that the experiments on cadavers showed only minimal laxity after section of this disk if the dorsal radio-

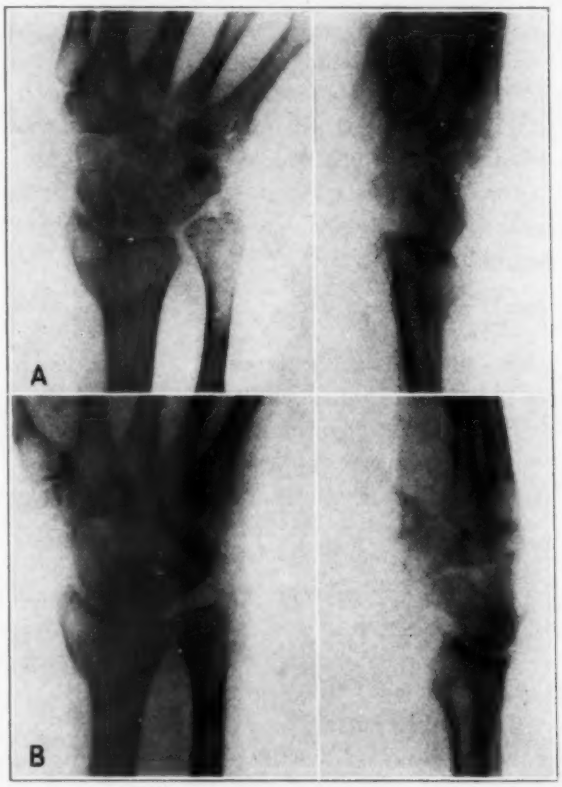

Fig. 5. (B. R.).—*A*, a roentgenogram taken after eight months of complete incapacitation following poor reduction of Colles' fracture. Motion of the fingers and extension of the wrist were full. Flexion was limited to 75 degrees and was slightly painful. Supination was limited but painless, and pronation was full but exquisitely painful. Tenderness was elicited over the head of the ulna, the styloid process of the ulna and the dorsal radio-ulnar ligament. There was free mobility of the ulna. *B*, a roentgenogram taken one month after suture of the ulna and excision of the styloid process of the ulna and the articular disk. All symptoms were gone except limitation of flexion.

ulnar ligament was intact. With the hand in pronation, the head of the ulna is pressed tightly against the sigmoid cavity of the radius, and the remaining dorsal radio-ulnar ligament and capsule are sewed

firmly together with fine catgut. Sufficient ligamentous tissue is always available for this purpose, and enough may remain to plicate and reenforce the suture line. If the styloid process of the ulna is displaced, the incision in the skin may then be laterally retracted and the styloid process excised. In this event, a firm suture is inserted to anchor the internal lateral ligament to the base of the head of the ulna. The incision is then closed, and a light circular dressing is applied. Some function may be permitted at the end of a week and, as a rule, in from three to six weeks symptoms have completely disappeared (fig. 5).

Subperiosteal resection of the head of the ulna as recommended by Darrach and Dwight is a valuable procedure, but its employment should be limited to those cases in which there is gross bony distortion.

In case of doubt the joint surfaces can be exposed and inspected through the approach described. If resection is then considered advisable, the incision can be easily extended for this operation. As yet, I have not had occasion to resect.

SUMMARY AND CONCLUSIONS

Symptoms referable to the radio-ulnar joint and pain on the ulnar aspect of the wrist joint are a frequent source of incapacitation following Colles' fracture and are almost invariably found in association with laxity of the radio-ulnar joint.

Laxity of the radio-ulnar joint and its consequent symptoms often persist, even after perfect bony replacement.

Rupture of the articular disk alone does not produce sufficient laxity to account for this condition (experiment on a cadaver).

The laxity must be due to a sprain or tear of the dorsal radio-ulnar ligament plus partial or complete rupture of the articular disk or its equivalent, avulsion of the styloid process of the ulna (experiment on a cadaver).

Laxity of the radio-ulnar joint may be diagnosed by manual examination immediately after reduction of the fracture of the radius (roentgenographic evidence is unreliable).

In the event of this finding, the position of supination offers the best opportunity for these structures to heal without tension, so preventing residual laxity of the radio-ulnar joint.

Mild symptoms may be considerably relieved by a broad laced leather wristlet.

A simple operative procedure is presented for the cure of laxity of the radio-ulnar joint and its attendant symptoms, in which any residual displacement of the radial fragment is not disturbed.

TORSION OF THE PEDICLE IN OVARIAN TUMORS

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Torsion of the pedicle is a serious and most common complication of ovarian tumor. The treatment in all instances is surgical. Ninety-seven cases of torsion of the pedicle were found at 1,101 consecutive operations for ovarian tumor performed at the Mount Sinai Hospital between 1924 and 1935. The grave hazards of this condition, the pathologic changes and the symptoms are of particular interest and importance. There is a dearth of literature pertaining to the subject.

Percentage Incidence of Torsion of the Pedicle in Various Types of Ovarian Tumors

Type of Tumor	Series at Mount Sinai Hospital		Grotenfeldt's Series, Percentage	Frank's[1] Series, Percentage
	Number of Cases	Percentage		
Fibroma	7	29.0	29.0	21.0
Papillary serous cystadenoma	36	25.0	18.4	15.1
Dermoid cysts	33	17.5	17.2	8.3
Papillary pseudomucinous cystadenoma	8	13.0	....	....
Papillary pseudomucinous cystadenocarcinoma	1	9.0	....	....
Simple cysts	36	7.0	21.3	20.0
Papillary serous cystadenocarcinoma	3	2.0	....	....
Sarcoma	0	...	6.4	12.5
Carcinoma	0	...	4.0	2.7

The incidence of torsion in this series of ovarian tumors at Mount Sinai Hospital was 9 per cent. Eden and Lockyer found a frequency of 2 per cent; Frank,[1] 8.5 per cent, and Araya and Carbo, 10.22 per cent. S. Wiener reported 33 instances of torsion or a frequency of 12.26 per cent in his series of cases of ovarian tumor and Grotenfeldt, 83 instances, or a frequency of 15.2 per cent. The figures on the comparative incidence of torsion of the pedicle among various types of ovarian tumor are given in the accompanying table.

The solid and semisolid tumors show the greatest frequency of torsion. In the series of cases of ovarian tumor observed at the Mount Sinai Hospital there were 522 simple cysts, with torsion in only 7 per cent. This is at variance with the figures of Grotenfeldt and Frank.

From the Gynecological Service of Mount Sinai Hospital.

1. Frank, R. T.: Gynecological and Obstetrical Pathology, ed. 2, New York, D. Appleton and Company, 1931.

CAUSE, EXTENT AND DIRECTION OF TORSION

The exact cause of twisting is not known. Factors favoring rotation are a smooth tumor surface, solidity, a tumor of moderate size, a roomy pelvis and a relaxed abdominal wall. Large growths are not prone to turn. A thin, long pedicle, acquired probably by traction, turns more readily than one which is short and thick. Infection and malignancy produce adhesions early, which immobilize the tumor.

It is noted in the table that solid and semisolid tumors rotate from two to four times more frequently than cystic ones. The relative frequency of torsion of the pedicle among solid fibromas (29 per cent) is not usually emphasized because of the rarity of this growth. Nevertheless, the high incidence is significant. Benign papillary, solid and semisolid tumors, for example, also show a much higher frequency of torsion than simple cysts, in spite of the fact that the former are more prone to adhesions from irritation, inflammation or local metastasis. The specific weight, therefore, may be a factor in favoring the rotation of ovarian tumors.

Rotation has been attributed to falls, sudden exercise, muscular effort or trauma.[2] It may be precipitated by contraction of the abdominal wall, straining, coughing and movement of the intestines and bladder, as well as micturition or defecation. The laxity of the abdominal wall after childbirth predisposes to twisting. Postpartum changes in intra-abdominal pressure, due to emptying of the uterus, may cause torsion suddenly, whereas constant, minute growth impulses covering a long period may effect rotation slowly (Frank). Torsion has been ascribed to fetal movements and change in uterine position, and it is thought that the unequal growth and weight of the different cystic areas may influence the condition.

The extent of the torsion may vary from one-half to fifteen turns of the pedicle. The greatest number of coils in this group of cases was seven. It is likely that emergence from the true pelvis rotates the tumor 90 degrees. Usually a complete twist suffices to produce some circulatory changes and symptoms. However, many turns of a long, thin pedicle may remain asymptomatic.

The single complete twist is the most frequent. It occurred in 36 of 96 cases, or 37 per cent. Partial turns varying from one-half to a complete rotation were found in 15 instances. One and a half, two and three twists occurred in 3 instances, respectively. There were 4 tumors with four pedicle turns in each case and single instances of five and seven rotations of the pedicle.

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2. Graves, W. P.: Gynecology, ed. 3, Philadelphia, W. B. Saunders Company, 1923, pp. 446-447.

Occasionally the infundibulopelvic fold and the broad ligament, as well as the fallopian tube and the round ligament, are involved. There were 17 cases of twisted fallopian tubes, 8 instances of twisting of the infundibulopelvic ligaments and 1 case of twisting of the round ligament. Extreme torsion of the broad ligament occurred twice.

DIRECTION OF ROTATION AND SIZE OF CYST

According to Küstner's law, a tumor on the left side rotates from left to right and one on the right side rotates from right to left.[3] Eighty-five per cent of cases of torsion adhere to this rule (Pfannenstiel). The operative reports mentioning the direction of rotation indicate that this was true in 9 cases.

Restitution or spontaneous retorsion rarely occur. No instance of this phenomenon was observed, however.

There is no relation between size of a tumor and tendency to torsion. Although torsion of tumors the size of a grapefruit is frequent (52 per cent) enough to suggest an influence, the equal incidence in tumors of extreme size, namely, the size of a football or of an apple (24 per cent each), would contradict this assumption. In addition, the size has no relation to the number of pedicle turns.

POSITION OF TUMORS

Although ovarian tumor is equally frequent on both sides (44 per cent on the right side and 34 per cent on the left in 1,101 cases), torsion is twice as frequent on the right side as on the left. There was torsion of 67 tumors on the right side and of 32 tumors on the left side (69 and 31 per cent, respectively).

The relative frequency of the different ovarian tumors in given locations is as follows:

Fibroma occurs four times more often on the right side, but torsion is only twice as frequent on that side.

Torsion of papillary serous cystadenoma is three and one-half times more frequent on the right side than on the left, although the incidence of the growth is equal for the two sides.

Torsion of the simple cyst is two and a half times more frequent on the right side than on the left, although the cyst occurs only one and one half times more frequently on that side.

Torsion of a pseudomucinous cystadenoma is three times more frequent on the right side than on the left, though the incidence of the tumor is only one and one-quarter times greater on the right.

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3. Kaufmann, E.: Pathology for Students and Practitioners, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 2, p. 1565.

A twisted dermoid cyst is found one and one-half times more frequently on the right side than on the left, although this growth occurs equally often on each side. Eleven per cent of dermoid cysts are bilateral, but no instance of bilateral torsion was found.

Fifty-seven per cent of papillary serous cancers occur bilaterally; the remainder are equally distributed on the two sides. With this type of tumor, torsion is twice as frequent on the right side.

In the group of cases reported here not a single instance was found of rotation with tubo-ovarian cyst or endometriosis of the ovary—conditions conducive to marked adhesions.

Thus, regardless of the incidence of these individual tumors on the right side, torsion occurs more frequently on that side.

SYMPTOMS AND PATHOLOGIC CHANGES

The clinical appearance of torsion depends on the suddenness and extent of vascular obstruction.[2] The degree of pathologic change is not proportional to the number of pedicle twists. Constitutional symptoms vary with the pathologic alterations.

Torsion may be asymptomatic if the process is slow and gradual, if rotation is less than 180 degrees or if the pedicle is long and thin; also, when there is no impairment in the flow of blood or when the rotated tumor is secondarily fixed by adhesions or loose impaction in the pelvis. A small number of women in the cases reported here were completely asymptomatic, and no change except slight edema of both the pedicle and the tumor was observed. Clear pelvic fluid and delicate adhesions may be found at this stage. Fifty-eight per cent of the tumors showed no structural secondary changes, even though as many as six or seven pedicle twists were found.

Torsion produces symptoms if vascular compression of the ovarian vessels is moderate and augmentative over a long period. Venous return becomes partially obstructed. The uterus and adnexal structures, proximal to the point of rotation, become engorged, owing to the effect on the free anastomosis medially, while the distal structures show marked edema and enlargement. Further diminution of venous return may produce hemorrhage into a cyst or its wall. Trauma, necrosis or atrophy of the pedicle have caused detachment of the tumor. Collateral circulation through adhesions to the omentum, intestine, peritoneum or neighboring structures may maintain the existence and integrity of the tumor, and it may appear to be part of another organ. Micro-organisms permeating through adherent bowel may set up an abscess followed by peritonitis and sepsis.

Twenty per cent of the patients had torsion of the types described. They complained of mild colicky pain and nausea, with occasional

vomiting. Although attacks were recurrent over a three to six year period, none of the women were confined to bed and all were able to attend to their daily tasks. Constitutional symptoms were not reported.

Torsion may be sudden with severe symptoms. Acute onset was common in 80 per cent of the patients; 20 per cent of these, although many were previously not aware of the tumor, were in partial or complete shock. The remaining 60 per cent suffered acute symptoms. Agonizing colicky pain that is hard to relieve, nausea, vomiting, distention and diarrhea are the usual complaints. With abdominal hemorrhage, pains in the shoulders and chest are to be expected. The patient may suddenly, after the initial pains, become extremely distressed, apprehensive and prostrated. The abdomen becomes rigid, distended and sensitive. Rebound tenderness and signs of peritoneal irritations become manifest, while a rapid, thready pulse, fever, leukocytosis and a rapid sedimentation rate complete the clinical picture. Vaginal examination may or may not reveal a mass, since the abdominal rigidity may entirely mask the presence of the tumor.

The diagnoses of ileus, peritonitis and acute suppurative appendicitis or cholecystitis, renal colic or hemorrhage, intestinal obstruction and ruptured ectopic pregnancy were made in cases of this series. The sedimentation rate was rapid in 30 per cent (intracystic or peritoneal hemorrhage, rupture of a cyst, infarction or gangrene). In 11 per cent there was urinary urgency, and in 8 per cent, diarrhea and vomiting.

COMPLICATIONS

Complete vascular compression with thrombosis of the ovarian vessels produces marked symptoms. Pathologic complications were present in 48 per cent of the cases in this series. Infarction of the edematous wall of the cyst and solid portions of the tumor (10 per cent), as well as hemorrhage into the cavity of the cyst (30 per cent), are common. With extreme rotation, the infundibulopelvic fold (8 per cent) and the round ligament (1 per cent) as well as the fallopian tube (8 per cent) and the parametrium (8 per cent) become involved. The pelvis is occupied by a tense dark plum-colored mass, which is glistening or dull. Rupture at this time (6 per cent) may occur spontaneously in any of the twisted tissues, particularly in the pedicle, the cyst or an involved blood vessel. Free bloody fluid is fairly common (16 per cent). Bleeding into the pelvic cavity (6 per cent) with gravitation to the diaphragm (5 per cent) and consequent irritation of the phrenic nerve nearly always occurs, producing characteristic pain in the shoulder and chest. Rupture into the parametrium (3 per cent) may be followed by complete unilateral extraperitoneal extravasation of blood. The outlook for fatal hemorrhage is particularly alarming with torsion of a malignant ovarian

tumor. In 1 instance of rupture of a cyst 2,500 cc. of hemolyzed blood was removed; in another instance of marked infarction with a ruptured pedicle, 1,000 cc. of blood was found. Both of the patients were admitted to the hospital in shock. Suppuration, abscess formation, necrosis (8 per cent) and peritonitis (2 per cent) may result when intestinal micro-organisms enter the tissues.

Uterine torsion is rare and bilateral twisting is very uncommon. During sexual maturity (A. Strauss), profuse and irregular uterine bleeding may be induced by ovarian torsion. Endometrial hyperplasia has been reported in this condition in the menopause. During pregnancy this accident is extremely hazardous, owing to suppuration and fatal peritonitis. Von Franque [4] stated that 20 per cent of pregnancies, accompanied by twisted cysts are interrupted. Torsion, however, practically never complicates labor.[5]

Thus, the most frequent complications of torsion are hemorrhage within the tumor, torsion of the fallopian tube and infarction. Hemorrhage into the pelvis in most cases is the most serious complication in the early stage. Torsion of the infundibulopelvic or of the round ligament is not infrequent. Rupture of the tumor or pedicle is an important factor in cases of shock. Torsion of the broad ligament and rupture into the broad ligament occur infrequently; the latter condition presents a problem in gynecological diagnosis. Varicosities of the broad ligament was an incidental finding in 1 case. In the cases of advanced torsion, infection, necrobiosis, necrosis and peritonitis may set in. A small number of patients showed these pathologic changes. Subdiaphragmatic blood is usually found in all cases of hemorrhage.

Torsion of the normal ovary, although rare, occurs principally in children.[6] An uncommon complication occurs when a twisted ovarian tumor becomes incarcerated in an inguinal hernia sac, simulating strangulation. There were no such cases in the present series.

The subsidence of pain within two or three days may mean severance of the pedicle or gangrene of the tumor, with circumscribed adhesive or spreading peritonitis. Pain in the shoulders or chest occurs within twenty-four hours after rupture (5 per cent).

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4. Von Franque, cited by Graves.[2]

5. Green-Armytage, V. B.: Cases of Torsion of Fallopian Tube and Ovary During Pregnancy, J. Obst. & Gynæc. Brit. Emp. **36**:87, 1929.

6. Baron, C.: Torsion of Normal Ovary: Case of Reoccurrence of Rare Pathologic Condition, J. A. M. A. **102**:1675 (May 19) 1934. Finley, R. K.: Diagnostic Value of Uterosalpingography in Pre-Adolescent Ovarian Torsion, Surg., Gynec. & Obst. **52**:723 (March) 1931; Torsion of Normal Ovary and Tube with Rupture of the Ovary and a Review of the Literature to Date: An Addendum, Kentucky M. J. **30**:233 (April) 1932.

The complications of torsion found in various types of ovarian tumors are mentioned briefly as follows:

In 7 instances of torsion of fibroma 14 different complicating lesions were found. Hemorrhage within the tumor occurred 4 times (57 per cent) and free pelvic blood 3 times (43 per cent), and necrosis of the tumor was found (14 per cent). There were 2 instances each of torsion of the fallopian tubes and the infundibulopelvic fold (30 per cent) and 1 instance of torsion of the round ligament.

In 33 cases of torsion of a dermoid cyst there were 16 complicating lesions. The principal one, namely, rupture of the tumor, occurred 8 times (24 per cent). Infarction of a twisted fallopian tube was observed 4 times (12 per cent), and there were 2 instances each of free pelvic blood and intracystic hemorrhage.

In 3 cases of papillary serous cystadenocarcinoma with torsion there were 6 complications. Rupture of the tumor and free pelvic blood were found in 2 instances each (66 per cent). Necrosis of the tumor and a ruptured pedicle occurred once (33 per cent).

In 8 cases of papillary pseudomucinous cystadenoma with torsion, 7 complications were noted. Free pelvic blood was found 3 times (35 per cent), and there were 2 instances each of rupture and necrosis (25 per cent each).

Of 36 cases of papillary serous cystadenoma with torsion there was hemorrhage in 4 (11 per cent). Rupture and necrosis occurred twice (55 per cent each). Thus, there are 6 complicating lesions in this group of 36 cases.

In the single case of papillary pseudomucinous cystadenocarcinoma with torsion there was dilatation of the vein of the broad ligament.

MENSES

The menses were normal in 60 per cent of the patients; in 32 per cent there was hyperfunctional bleeding, namely, menorrhagia (20 per cent) and metrorrhagia (12 per cent), and in 4 per cent there was a hypofunctional menstrual condition, namely, a menopause without bleeding (4 per cent) and prepuberty amenorrhea (1 per cent). According to Graves,[2] the menstrual cycle may be altered by pelvic hyperemia induced by this condition. Dysmenorrhea was reported in only 8 of these cases.

CONCLUSIONS

1. Rotation is the most frequent (9 per cent) and dangerous complication of ovarian tumors. The treatment is always surgical.

2. Complications occur in 48 per cent of twisted tumors and depend on the degree of vascular compression. The extent of pathologic

changes and the severity of symptoms are directly dependent on the blood supply and the suddenness with which obstruction takes place.

3. The cause of torsion is unknown; factors of greatest influence are the specific weight or solidity of the tumor (75 per cent), sudden motion of the body and a tumor of moderate size (52 per cent) (the size of a grapefruit). Adhesions prohibit torsion.

4. The single complete twist is most common (37 per cent). The greatest number of pedicle turns in this series of cases was seven.

5. Ovarian tumors (1,101 cases) occur with almost the same frequency on both the right and the left side, yet ovarian torsion on the right side is twice as frequent as on the left.

6. Torsion may be asymptomatic, mild and recurrent, or extremely acute in onset with partial or complete shock. The corresponding pathologic changes of the tissue depend on the progressive or sudden vascular obstruction, varying from edema to infarction and hemorrhage.

7. Complications are rupture with hemorrhage, infarction, torsion of the fallopian tube and bloody or clear peritoneal fluid. These are in order of their frequency. Necrobiosis, gangrene and peritonitis are the end-results.

8. Torsion of tubo-ovarian cyst or of endometriosis of the ovary is not found.

9. The menses are undisturbed in most cases (60 per cent); hyperfunctional bleeding is the most frequent alteration among menstrual abnormalities. The incidence of dysmenorrhea is insignificant.

10. The diagnosis may be extremely difficult when the patient is examined in the acute stage. There is a high incidence of diagnostic error.

A REVIEW OF UROLOGIC SURGERY

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KIDNEY

Anomalies.—Stevens [1] reviewed a series of 27 cases of solitary pelvic kidney, consisting of 25 cases reported in the literature and 2 cases which he had observed. In 1932 Collins found 572 cases of unilateral renal agenesis and 49 other probable cases reported in the literature; he added 9 cases which he had observed personally. It therefore may be presumed that approximately only 4 or 5 per cent of single kidneys are true pelvic ones. Speaking in round figures, if one may expect a solitary kidney once in every 1,000 persons, there will be a solitary pelvic kidney in 1 of about every 22,000 persons.

The indications for the treatment of ectopic kidney, surgical or otherwise, are in general governed by the well known urologic principles employed when the organ is normally situated. However, because of the relatively poor drainage and mild hydronephrosis universally present in an ectopic kidney and because of the irregular and frequently multiple blood supply, nephrectomy is more commonly demanded (when there is present elsewhere in the body a normal kidney). The blood supply of a pelvic kidney is variable; it comes from the bifurcation of the aorta, the common iliac arteries, the external or internal iliac arteries and the median sacral and other neighboring vessels. The veins usually correspond to the arteries.

1. Stevens, A. R.: Pelvic Single Kidneys, Tr. Am. A. Genito-Urin. Surgeons **29**:403-413 (May) 1936.

In the series of 27 cases of solitary pelvic kidney reviewed here, satisfactory descriptions of the blood supply are few. In 3 cases, including the case reported by Stevens in which surgical treatment was employed, the vessels supplying the kidney apparently came from the region of the bifurcation of the aorta only (none entering the kidney laterally), which made possible a free manipulation of the kidney for conservative measures. In 16 of the cases in which the sex was stated, 5 of the patients were males and 11 were females. The ages varied from a fetus up to 48 years. Calculus was noted 3 times, and successful operation was performed in 2 cases. In 14 cases there was some abnormality or deficiency of the sexual apparatus, which is a common finding. Many of the observations were made at necropsy, but others were made clinically in cases in which no treatment of the urinary tract was indicated. Only 11 of the patients had undergone some operation which concerned the renal condition. In 2 cases the single kidney was removed together with a neoplasm or because one was thought to be present, with, of course, subsequent anuria and death. Exploratory laparotomy was performed in 4 cases. An appendectomy was performed in 1 case. In another, the kidney, which had not been recognized, was punctured, and tissue was removed for diagnosis; in another case the kidney was mistaken for the uterus and opened, but was promptly closed. Nephrostomy was performed once and lithotomy twice; all of these operations were successful.

The gross errors were noted chiefly in earlier publications. The prevalent general use of cystoscopic and roentgenographic investigations supplies ample data to forewarn and guide the surgeon.

Gutierrez [2] stated that all anomalies of the urinary apparatus are produced by malformations of embryonic origin. All such anomalies are pathologic and are potentially surgical. Of all the anomalies that come under clinical observation, those of the kidneys and their pelves and ureters are the most common.

The table shows the multiplicity of the different types of malformation that are encountered by the urologist. The variation in the symptoms that accompany these anomalies results in many errors of diagnosis and sometimes in unnecessary operations. Pathologic conditions are more likely to be found in kidneys and ureters that are congenitally abnormal than in those that are perfectly normal. More often than not congenital malformations of the upper part of the urinary tract produce urinary retention and infection, dilatation and loss of function of the kidney, pelvis and ureter.

Anomalies of the upper part of the urinary tract fall into two classes: those that are potentially surgical and those that present definite patho-

2. Gutierrez, Robert: Sur les anomalies pyélo-urétérales et leurs conséquences chirurgicales, Cong. franç. d'urol., Paris, 1936, pp. 194-214.

logic changes. There is an enormous amount of diversity in the diagnostic and therapeutic problems presented by such kidneys. They should, however, be regarded as clinical entities, and their treatment should be chosen individually. In diagnosis, prognosis and treatment, an indispensable part is played by urography. When all renal function has been destroyed by pathologic processes, intravenous pyelography

Classification of Pyelo-Ureteral Anomalies

- **Calices**
  - Absent
  - Ectopic
  - Abnormal in number, size or position
- **Pelvis**
  - Absent
  - Rudimentary
  - Misplaced
    - Excessively rotated
      - Anterior
      - Posterior
    - Not rotated
  - Double (bifid) or multiple
    - Unilateral
    - Bilateral
  - Hydronephrosis (congenital)
    - Pelvis intrarenal
      - Unilateral
        - Total
        - Voluminous
        - Partial
      - Bilateral
    - Pelvis extrarenal
  - Diverticulum of pelvis
- **Ureter**
  - Anomalies of number
    - Single (one ureter absent)
    - Double on one side
    - Triple
    - Quadruple
      - Complete
      - Incomplete
    - Quintuple
    - Sextuple
  - Anomalies of form, size and position
    - Diverticulum of ureter
      - Simple
      - Multiple
    - Ureter imperforate
      - Pyo-ureter
      - Hydro-ureter
    - Stricture of ureter
      - Pyelo-ureteral
      - Vesico-ureteral
      - Median
    - Dilatation of ureter
      - Total: megalo-ureter
      - Partial: hydro-ureter
    - Golf-hole type, with reflux
    - Ureter
      - With ectopic orifice into bladder
        - At fundus
        - Into a diverticulum
        - Near neck of bladder
    - Ectopic
      - With extravesical orifice opening into
        - Other ureter
        - Urethra
        - Genital tract
        - Intestine
    - Crossed ureters
      - With renal fusion
      - Without fusion
      - Unilateral transverse
      - Bilateral transverse
    - Ureter twisted or occluded
    - Postcaval ureter
    - Ureter with valve formation
    - Ureterocele

is useless for diagnosis, but it should nevertheless always be employed when ureteral catheterization is impossible. In cases of ectopic ureter, chromocystoscopy facilitates the preoperative diagnosis.

Treatment may be regarded as either urologic or surgical. The former is of importance for the treatment of infection and for the purpose of assuring satisfactory drainage. Surgical treatment may be either conservative or radical, according to the type of pathologic con-

dition that is found. The results of surgical treatment are likely to be more satisfactory if the diagnosis is made early, as soon as the first symptoms arouse a suspicion as to the patient's condition.

If the function of the kidney is still satisfactory, it is possible in some cases to do a plastic operation to correct the deformation of the excretory passages. But if for lack of drainage the kidney has become hydronephrotic and has been to a large extent destroyed, nephrectomy will be necessary, provided the kidney on the opposite side has adequate function. It is evident that an anomaly of the renal pelvis may produce a urinary stasis that not only will affect the pyelo-ureteral tree but will result in complete destruction of renal function. The commonest anomaly of the pelvis of the kidney is hydronephrosis; it may be due to aberrant vessels associated with kinking of the ureter and nephroptosis. Under such conditions, conservative treatment will consist of ligation of the vessels, followed by nephrolysis, ureterolysis and nephropexy, especially if the hydronephrosis is small and the renal function sufficient. In the presence of a large congenital hydronephrosis with complete destruction of renal function, radical operation is the only recourse, if the condition of the other kidney permits. Ureteronephrectomy is indicated in cases in which both hydronephrosis and hydro-ureter are present, due to congenital malformation of the lower part of the ureter, such as an intramural stricture of a "golf-hole" orifice with vesico-ureteral reflux on one side. On the other hand, when a small congenital hydronephrosis exists as the result of pelvic retention due to lack of tone and stricture of the ureter, it may be sufficient to carry out cystoscopic treatment, with dilation of the ureters by bougies and lavage of the renal pelvis.

In certain cases in which a plastic operation is indicated, such as reconstruction or resection of the pelvis for hydronephrosis, the carrying out of a pyelo-ureteral anastomosis or transplantation of a ureter, and in cases in which it is an advantage to free the ureter from bands of adhesions and blood vessels, it may be wise first to do a nephrostomy in order to divert the urine and assure temporary drainage. This will relieve the pelvis from hydraulic pressure as well as from infection and will assure not only the restoration of good function but a sound prognosis.

In a series of 97 cases of ectopic kidney reported by Thompson and Pace,[3] the condition was discovered at necropsy in 9 cases and by physical examination or surgical exploration in the 88 remaining cases. This is an incidence of about 1 in every 1,200 necropsies performed at the Mayo Clinic and represents an incidence of approximately 1 in every 10,000 patients observed clinically. Because of the lack of symp-

3. Thompson, G. J., and Pace, J. M.: Ectopic Kidney: A Review of Ninety-Seven Cases, Surg., Gynec. & Obst. **64**:935-943 (May) 1937.

toms which might lead to urologic investigation, renal ectopia is often discovered only at the time of surgical exploration; in 8 cases in this series the ectopic kidney was a congenital solitary organ, which fact must be carefully weighed if any one contemplates nephrectomy in cases of renal ectopia without the advantage of preoperative urographic study. Twenty-nine, or 32.9 per cent, of the 88 patients who clinically were found to have ectopic kidney suffered from recurrent pain, which indicates that the ectopic kidney as a rule drains poorly, owing either to its position or to constriction of the ureteropelvic juncture by anomalous blood vessels or perirenal tissues.

Treatment such as ureteral dilation may cause harm and generally will not benefit the patient. As a general rule, if treatment is indicated, it should be surgical. Conservative operations, such as plastic procedures on the renal pelvis or ligation of anomalous vessels, are likely to fail. Nephrectomy is therefore the treatment of choice if the function of the opposite kidney will permit. The only exception is pelviolithotomy in cases in which there is little or no hydronephrosis. Pregnancy will generally be uneventful and labor normal in spite of an ectopic pelvic kidney.

Pilcher and Braasch [4] said that three anomalies of renal development which may be confused are hypoplastic kidney, renal aplasia and congenital absence of one kidney. Such malformations of the upper part of the urinary tract represent failure of proper development during embryonic life and must be clearly distinguished from atrophy of the kidney, which may follow trauma, tuberculosis and other infections, and hydronephrosis, calcareous disease or ureteral obstruction from any cause.

A hypoplastic kidney is usually from a third to a sixth the normal size, and its mate is correspondingly hypertrophied. Except for its small size it may be normal anatomically and histologically. The ureter is always patent, and excretory function may be good in regard to the elimination of urea and dye. In renal aplasia the rudimentary structure is represented by a small mass of fibrous tissue, which histologically may be composed of embryonic glomeruli and tubules in various stages of degeneration. There is no true pelvis. Congenital absence of one kidney means complete lack of development of one of paired organs. There is complete absence of the kidney, ureter and the part of the trigon on the same side. The diagnosis of these malformations is made by cystoscopic examination and intravenous urography.

A 27 year old man complained of painful, frequent micturition and pain in both flanks. Dye tests revealed a functionless right kidney. Nephrectomy was performed, and a typical hypoplastic kidney with

4. Pilcher, Frederick, Jr., and Braasch, W. F.: Hypoplastic Kidney: Report of a Case, Proc. Staff Meet., Mayo Clin. **12**:52-56 (Jan. 27) 1937.

moderate pyelonephritis was removed. The patient's symptoms were completely relieved.

De Quevedo [5] pointed out the great frequency of polar arteries and the important part they play in the blood supply of the kidney. He observed such arteries in 19 of 30 cadavers; in 13 they were at the upper pole, and in 6 they were at the lower pole. In this study he considered as polar arteries only those supernumerary arteries which, starting at a distance from the principal trunk of the renal artery, come from the aorta and disgorge at one of the poles, and arteries which come from the renal artery at a point prior to its ramification and are directed to one of the poles. He was not concerned with normal ramifications at the hilus which are toward the poles.

In order to discover how large a portion of the renal parenchyma is supplied by such abnormal polar arteries, de Quevedo inserted into the arteries of cadavers a paraffined glass tube to which he attached a pump and a mercury manometer. Forcing water into the kidney by this route, he found that when the pressure reached 6 cm. of mercury the water passed out through the renal vein and in part through the ureter. In the course of half an hour, under constant pressure, there appeared outlined on the renal parenchyma, which was red from stasis of blood, a pale or white zone, of irregular circular contour and varying size, which indicated approximately the superficial territory of the parenchyma corresponding to this vessel. Then, to discover how far this zone extended into the interior of the kidney, he used the method of corrosion and injected substances of different colors into the polar artery and the renal artery, so that he could easily determine with accuracy the extent of renal substance belonging to each vessel. The results showed that in some cases the abnormal polar artery supplied renal substance corresponding approximately to a fourth or even a third of the entire kidney.

In view of this fact, de Quevedo suggested that in conservative operations on the kidney, whenever it is necessary to exteriorize the organ, an attempt should be made, when possible, to avoid the destruction of such an artery for the following reasons: (*a*) A portion of the parenchyma is lost through an infarct; (*b*) secondary hemorrhage is possible after infection of the infarct; (*c*) suppuration of the infarct, if it occupies a calix, may cause a postoperative urinary fistula and infection of the wound, and (*d*) in an operation like nephrotomy the amount of functioning parenchyma will be greatly reduced if the portion lost through section of the polar artery is added to the portion lost by incision.

5. de Quevedo, Angel: Importance des artères polaires dans la chirurgie conservatrice du rein, Arch. d. mal. d. reins **10**:269-281 (May) 1936.

It appears from a study of the literature on the subject that surgeons have not given attention to these eventualities and have not made postoperative functional tests to compare the capacity of the kidney the polar artery of which has been tied with that of the other kidney. It seems evident that when such an artery is of large caliber the functional loss entailed must be considerable.

Tumors.—Barringer [6] stated that there are many interesting data both in the literature and in the case records at the Memorial Hospital which shed a good deal of light on the effects of irradiation of tumors of the kidneys.

Investigation showed the large Wilms tumors of children to be as radiosensitive as metastatic teratomas and to undergo the same remarkable primary regression. The regression seemed to be temporary, however. The tumors grew again after perhaps a few months. When again irradiated they did not recede as readily as at first. If irradiation therapy alone was used, the disease proved fatal.

Priestley and Broders considered results of nephrectomy alone in the treatment of Wilms' tumor and reported 2 cases in which it was used; 1 patient lived thirteen years and two months, and 1 lived three years and three months, respectively, after the operation. These 2 cases are from a series of 65 cases of Wilms' tumor, in 20 of which nephrectomy was performed. One of the authors' conclusions was as follows: "The group who were treated by irradiation and nephrectomy survived approximately twice as long as the group who were treated by operation or irradiation separately. No patient to our knowledge has been cured of Wilms' tumor by irradiation alone." Alcock, in a personal communication to Barringer, stated that among nephrectomized patients the mortality in the first eighteen or twenty-one months was just 100 per cent. Other authors reported that 90 per cent of children die in the first year after operation. The operative mortality is between 15 and 30 per cent. In a large number of cases the lesion is inoperable.

In consideration of the results of irradiation followed by operative removal, Randall has reported several cases, but in none of them did the patient survive long after nephrectomy. Alcock reported a tumor of the kidney in a girl 3½ years of age. The tumor was irradiated for six weeks, and the kidney was removed. The child was alive and well approximately three years after she was first seen. Barringer had 2 patients, 1 of whom died six months after admission and four months after an easily performed nephrectomy. The record in cases in which a tumor of the kidney has been removed after irradiation is not brilliant. The life span of the patients has apparently

6. Barringer, B. S.: Radio-Sensitive Kidney Tumors, Tr. Am. A. Genito-Urin. Surgeons **29**:415-427 (May) 1936.

not been much lengthened. Unquestionably, the operative hazard is less if the kidney becomes smaller after irradiation. Barringer stated that surgeons have been too impatient to remove the tumor after irradiation. Ewing has emphasized this. As far as one can see from case records, most surgeons have so indulged themselves. Barringer expressed the belief that the time of election for removal of such a tumor is when it has reached its maximal regression after irradiation, which generally requires from one to two months. He was convinced that irradiation should be continued somewhat longer and that operative removal should be deferred as long as possible.

Cutting into a tumor composed of highly malignant cells would seem theoretically at least to be perilous. In 4 of Barringer's 10 cases, because of squeezing the kidney or cutting or breaking into it at the time of operation, there were rapid local recurrences. In 1 of these cases there had been inadequate preoperative irradiation of the kidney. One could not help coming to the positive conclusion that incision of such a tumor for the purpose of diagnosis should not be done. If the kidney is not removed, one should be content to rest the diagnosis on the pyelogram, the tumor and its reaction to irradiation.

Theoretically, the trauma resulting from the exploration, the attempt at removal or the actual removal of a Wilms tumor is an ideal method of releasing tumor cells, which enter the blood stream and cause distant metastasis. In 6 of Barringer's 10 cases there was distant metastasis; 4 patients had undergone nephrectomy, 1 had been subjected to nephrotomy and 1 had not had any operation. In the 4 cases in which there was no distant metastasis 4 of the patients had undergone either nephrectomy or nephrotomy. These cases are too few to give much support to this theory. Transperitoneal nephrectomy is the operation of choice and should prevent metastasis in a certain percentage of cases.

In Barringer's series, as measured by the pathologic reports on removed and irradiated kidneys and by end-results, sufficient irradiation to control the primary tumor was not or could not be given. By observing the effects of irradiation on children, one is impressed with two reactions. Apparently, the skin of children stands irradiation better than that of adults, but the general condition of children is more affected. In particular, leukopenia develops; the hemoglobin content and the number of erythrocytes remain the same or even increase. Rarely, irradiation causes severe and even fatal bleeding. There was 1 such case at the Memorial Hospital. Barringer has been able to collect from the literature and the records of the Memorial Hospital reports of 6 cases in which irradiation alone has apparently controlled the disease.

Kirshbaum and Culver [7] reported 2 cases in which sarcoma originated in the region of the renal hilus: a fibrosarcoma and a mixed tumor with sarcomatous elements. The true character of the neoplasm in each instance was not suspected before operation. The diagnosis was made from the pathologic examination of the excised specimens. Clinically, this condition presents vague symptoms, and the diagnosis is usually made very late. It may be well to remember that a tumor found compressing the kidney may have its origin in the renal hilus, which may help to explain the origin of some of the obscure sarcomas situated either in the perirenal or in the retroperitoneal tissue.

Roth and Davidson [8] reported a case of pulsating metastatic hypernephroma of the sternum. This case is unique in that an additional primary neoplasm was present in the brain. Primary pulsating neoplasm of the sternum has not been reported. Cases have been reported of pulsating neoplasm of the sternum which was secondary to hypernephroma or to malignant adenoma of the thyroid gland. In the absence of an aortic aneurysm and demonstrable neoplastic changes of the thyroid gland, a pulsating sternal mass should be strongly suspected to be a metastatic hypernephroma, even though clinical evidence of a primary renal tumor is wanting. An apparently solitary metastatic hypernephroma of the sternum is likely to be accompanied by silent, undetected metastasis elsewhere, and surgical intervention is therefore inadvisable.

Perrier [9] reported a case of carcinoma of the pelvis of a horseshoe kidney in a girl aged 4 years. The first symptom was a violent attack of hematuria, unaccompanied by any other signs of renal disturbance. Urinalysis done three times in the course of ten days showed sterile hematuria, with no further changes other than the presence of a few hyaline cylinders. Catheterization revealed that the blood came from the right kidney. Careful palpation revealed nothing abnormal, and the child played, ate and slept as usual. When chromocystoscopy and roentgenography both gave negative results, Perrier was compelled, by exclusion, to consider the idea of a tumor of the right renal pelvis, notwithstanding the rarity of such a condition in a child.

A lumbar operation, performed two months after the first sign of hemorrhage, revealed the presence of a horseshoe kidney and a soft tumorous mass in the right side of the pelvis. The two kidneys were united at the lower poles by an isthmus of true renal parenchyma. Perrier divided the isthmus and removed the entire right half of the

7. Kirshbaum, J. D., and Culver, Harry: Sarcoma of the Renal Hilus, Surg., Gynec. & Obst. **64**:944-948 (May) 1937.

8. Roth, L. J., and Davidson, H. B.: Metastatic Pulsating Tumors of the Sternum Secondary to Renal Hypernephroma, J. Urol. **37**:480-489 (April) 1937.

9. Perrier, M.: Carcinome du bassinet d'un rein en fer à cheval, J. belge d'urol. **5**:357-363 (Oct.) 1936.

horseshoe organ. Operation was followed by prompt recovery, and the child was dismissed in good condition twelve days later.

The pathologic examination revealed within the pelvis of the kidney a friable neoplastic mass which microscopically had the character of a carcinoma of the renal pelvis. It had started at the free pole, at the level of a flattened papilla, where it became integrated with the point of the pyramid; here, it had the form of a pavement epithelial growth, but as it advanced farther from its starting point it gradually lost its alveolar structure and finally became a solid carcinoma, thus presenting a rather unusual type of architecture.

The prognosis of these tumors must be reserved, especially in young children, as recurrences are common even after three or four years. This case is of special interest etiologically, in view of the presence of a horseshoe kidney, which points to the existence of a congenital anomaly of the calix, in which it had its site, as the original cause of the neoplasm.

Dean [10] discussed a few of the fundamental factors concerned with preoperative irradiation in the management of renal tumors.

Realizing the diagnostic importance of a histologic examination and hoping that by its aid an early differentiation between radiosensitive and radioresistant tumors could be obtained, Dean has obtained biopsy specimens by aspiration from all suspected renal tumors for the last eighteen months. The technic is not difficult, and no patients have been harmed as far as he knows. After examining the smears a skilled pathologist can make a positive or negative diagnosis of tumor, but grading has been impossible and predictions regarding radiosensitivity cannot be made.

Preoperative irradiation of renal tumors has been recommended to devitalize tumor cells and thereby lessen the likelihood of disseminating metastasis by operative manipulations. Little proof exists that metastasis is promoted by careful operation. Furthermore, if it were a common occurrence it is doubtful whether preoperative irradiation could completely eliminate the danger. Even in those tumors in which the most marked regression has been obtained by irradiation, one can usually find a certain amount of apparently viable tumor tissue. However, until there is more exact knowledge on this point it is more conservative to reduce the possibility of metastasis in any practical way.

Preoperative irradiation of renal tumors has also been recommended to diminish the size of the tumor and make nephrectomy easier. This is an important effect of irradiation and is of much clinical importance. A large proportion of the tumors of the renal parenchyma in adults and the great majority of Wilms tumors in children shrink when irradiated. Only a therapeutic test will determine how much an individual tumor

10. Dean, A. L., in discussion on Munger, Arbor D.: Irradiation of Malignant Renal Neoplasms, J. Urol. **37**:692-694 (May) 1937.

will respond, but the benefit may be so marked that the preoperative irradiation of all large renal tumors is recommended.

Preoperative irradiation of renal tumors has also been recommended to make inoperable tumors operable. In this discussion an uncomplicated primary tumor was not classified as inoperable because of its large size alone.

It is important to know the amount of radiation which can be delivered to the average renal tumor. With a voltage of 200 kilovolts to the anterior, posterior and lateral portals, each as large as the size of the patient's body permits, at a target-skin distance of 70 cm., and in divided doses of 300 roentgens to one portal daily, filtered through 0.5 mm. of copper, it is possible to give most patients 2,400 roentgens to each portal in about twenty-six days. In treating infants, 200 roentgens per day at a distance of 50 cm. has been effective. No attempt should be made to give a predetermined total dose to these small patients. Careful examinations should be made before each treatment. The effects of irradiation can best be gaged by the clinical changes noted from day to day.

The value of a preoperative cycle of irradiation which may deliver a maximal dose of 5 threshold erythema doses to a tumor depends largely on the cellular make-up of the growth. Epithelial tumors of the renal pelvis are radioresistant. For this reason and because they tend to remain localized for a long time and because obstruction and infection usually are present, preoperative irradiation is not recommended. At the opposite extreme of radiosensitivity are Wilms tumors, which usually are composed of embryonal tissues. Because these tumors are large when discovered and because they usually regress readily under irradiation, preoperative treatment is of the greatest value. Tumors of the renal parenchyma in adults generally are of an intermediate radiosensitivity, but there may be considerable differences within the group. Preoperative irradiation is recommended in these cases. With the present inadequate knowledge and until more powerful sources of irradiation are provided, it is probably safer to regard preoperative irradiation strictly as an adjunct to operation.

If a growth is radiosensitive, regression will be prompt and rapid. Perhaps the tumor will disappear after only a part of the maximal dose of irradiation has been given. Since this is as much regression as can reasonably be expected, nephrectomy can be performed without further delay. For a tumor which is more radioresistant it may be necessary to give a full dose of radiation and wait a few weeks before the greatest possible regression takes place. However, little is to be gained by a delay of more than a month.

Dean has found that healing of nephrectomy wounds may be delayed by heavy irradiation, but so far this disadvantage has been of relatively little practical importance.

Jasienski,[11] on the basis of a case which he observed, pointed out the ease with which one may mistakenly diagnose, by means of pyelography, a hydronephrosis that does not exist. In this case it was believed that the patient was suffering from a hypernephroma of the right kidney, complicated by hydronephrosis. The patient died in a few days, and necropsy revealed that the right kidney was not enlarged but that it actually was smaller than the left one, which was healthy. Its position, however, was abnormal, which had led to misinterpretation of the pyelogram. On its upper surface was an enormous hypernephroma, and around it there were several other solid tumors, above, below and outside of the organ. These growths had crowded the kidney out of its proper position and had pushed it toward the median line and the anterior ventral wall. The majority of surgeons are satisfied to make a single pyelogram with the patient in the dorsal position, which ordinarily gives a correct view of the positions of the calices, pelvis and ureter. But this case shows that under certain conditions the image of the pelvis filled with opaque fluid as it is seen in the roentgenogram may not be in accordance with the true state of affairs and may lead to an erroneous diagnosis. It teaches that pyeloscopy is the only method that can prevent such errors, since it permits the exploration of the image of the renal pelvis under the roentgenoscope and in all directions, and that hydronephrosis can never be confused with a shadow due to an abnormal position of a kidney. In the pyelogram, however, since the tumor had displaced the kidney, the shadow of the renal pelvis was much larger and much farther from the median line than the organ was in reality. Only pyeloscopy, or else a second pyelogram taken with the patient in the ventral position, would have been adequate to prevent the erroneous diagnosis that was reached. In this case, owing to the fact that the pelvis was dilated and that it constituted in any event a sound reason for operating, the failure to make an exact diagnosis had no adverse effect on the patient's welfare, but the experience shows the greater exactitude of pyeloscopy as compared to pyelography.

Stone.—Randall [12] offered a preliminary report to prove that a renal stone originates by growth on an initiating lesion. Secondarily, the fact that such an initiating lesion must be sought for on the renal papilla has, in his opinion, likewise been proved in the microscopic work performed and by the findings related, for he has been fortunate in the material available, as he found rather typical progressive steps in the growth of a calculus. He also described a pathologic process occurring in the wall of the renal papilla which hitherto has not been recognized and which consists of a subsurface deposit of calcium.

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11. Jasienski, G.: De la radiographie des voies urinaires dans les tumeurs solides du rein, J. d'urol. **42**:543-548 (Dec.) 1936.

12. Randall, Alexander: The Initiating Lesion of Renal Calculus, Tr. Am. A. Genito-Urin. Surgeons **29**:323-330 (May) 1936.

In the series of cases presented Randall stated that he had traced from the subsurface deposit of calcium the destruction of its covering surface membrane and the deposit thereon of two characteristic types of urinary salts—calcium phosphate and calcium oxalate. These salts appear to be direct secondary deposits on this calcium plaque once it is denuded of its epithelial covering. It was his belief that once such a calcium plaque has lost its covering membrane and is bathed in the urinary excretion, there then are deposited on this plaque, as a foreign body, certain salts normally present in the urine, and the salt so deposited follows closely the well recognized laws of the crystallization of salts from any solution wherein seeding for crystallization and supersaturation play the dominant rôles and are the determining factors as to the character of salt to be deposited. He did not presume that the described pathologic stages constitute the sole initiating lesion on which all stones originate but reserved for further study the possible relationship of focal infection, septic and aseptic ulceration, infarction, allergic reactions of mucous membrane and keratinization from avitaminosis.

Gabrielle and Girgensohn [13] stated that urinary stasis, whether septic or aseptic, is undoubtedly one of the most important factors in the pathogenesis of renal lesions. While there is a large literature concerned with the results of aseptic stasis, there is little knowledge of the mode of propagation of inflammatory processes of pyelic origin, since the relation between urinary stasis and ascending infection has not yet been ascertained.

The authors undertook experiments with the injection of india ink into rabbits, with a twofold purpose: (1) to ascertain the mode of diffusion of the nonbacterial contents of the renal pelvis during the course of an exaggerated pressure exerted on them by stasis of various types and duration and (2) to determine the routes of ascending infection and, for each route, the specific changes occurring in the renal parenchyma. In the second series of experiments an infection of the renal pelvis was provoked in the course of these various types of urinary stasis.

It was found that an acute increase of pressure within the renal pelvis causes rupture of the fornix, associated with injection of the lymphatic and venous systems. But rupture does not occur when as a result of a long-standing chronic urinary stasis the fornix is able to assume a rounded form and hydronephrotic atrophy becomes manifest. These anatomic modifications can be found at the end of the first week after ligation of the ureter or at the end of the second week after artificial production of a ureteral stenosis.

13. Gabrielle, S., and Girgensohn, H.: L'influsso della stasi urinaria sulla diffusione nel parenchima renale del contenuto pielico asettico e settico, Arch. ital. di urol. **13**:519-547 (Sept.) 1936.

With the appearance of this hydronephrotic atrophy, pyelovenous reflux and injection of the lymphatic system cease simultaneously. There is not enough rounding of the fornix in acute dilatation to prevent its rupture. It is only in chronic urinary stasis due to hydronephrotic atrophy that stabilization of the modification of the form of the fornix is achieved and renders it capable of successfully withstanding the increased pressure within the renal pelvis. It was possible in such a pelvis to reach a degree of pressure that would overcome the mechanism of closing of the papilla and cause an injection of the tubules, which increased as the dilatation of the pelvis went on. It was of interest that in a certain group of kidneys in which there were cicatrices of the cortex of unknown origin the spontaneous changes observed were sufficient to cause evident injection of the tubular system accompanied by prompt rupture of the fornix; partial modifications of the symmetry of the fornix also led in the same direction.

In the second series of experiments it was observed that ascending infection is diffused as follows: In the normal kidney infection rarely finds an entry. But when there is pyelitis, the infection is diffused almost exclusively to the lymphatic system through the fornix. This lymphogenous propagation is present in all degrees of urinary stasis, up to the point where the fornix disappears through the effect of the progressive hydronephrosis.

In hydronephrosis the phenomena of lymphangitis are totally masked by the rapid propagation of the bacteria into the urinary canaliculi. Within a few hours the lymphangitis has reached the renal cortex, where it immediately causes inflammation. But it is only the association of bacteria which arise in the renal pelvis with those that are eliminated from the cortex by means of the urinary flow that governs the occurrence of grave inflammation and necrosis of the medullary cone. These changes dominate the disease picture of the last stages. The more the hydronephrotic atrophy progresses, the more impervious do the canaliculi and the lymphatic vessels become; they cause the infection to slow up and to progress with more and more difficulty.

If in a normal kidney an infection of the pelvis is produced simultaneously with an acute urinary stasis by the tying of the ureter, rupture of the fornix occurs on the second or third day afterward. Diffusion of the contents of the renal pelvis then causes an abscess of the hilus of the kidney, which is accompanied at the same time by a rapid spread of the infection through the tubules and, more slowly, by a spread through the lymphatics. Only in the initial stages, however, can the individual modes of propagation be recognized, some one of which is generally found to predominate over the others. In the later stages it is not possible to establish any distinction between the several routes by which the infection has been disseminated.

McCague [14] reported a study of the problem of traumatic renal and vesical calculi.

Stones form in stone-free patients who are immobilized for long periods because of fractures of the long bones, lumbar vertebrae and bony pelvis and also because of osteomyelitis. This is an important clinical problem, as grave injury may take place; in some instances it may result in urosepsis and death. In all cases the following factors are constant: urinary stasis, urinary infection, alkaline urine and the stones, which are phosphatic. A plan for the prevention of the formation of this type of stones was suggested.

The major problem is that of prevention. In all cases of fracture patients who are potentially liable to have stones are carefully studied as to their status on admission. The character, bacteriology and p_H of the urine are noted, Jean's test is performed, the urinary tract and the bony skeleton are examined roentgenographically and quantitative tests are made for serum calcium, phosphorus, protein and phosphatase. In this group of cases rigid measures to prevent the underlying causes of the formation of stones are put in operation at once.

If the patient is immobilized in a fracture bed, frequent elevation of the renal level is made to combat stasis by changing the position consistently with the treatment of the particular fracture. Extreme care is used in any instrumentation that may be necessary. Elimination of any foci of infection is effected when possible. The patient is immediately placed on an acid ash diet with an excess of acid ash of from 20 to 30 cc., dependent on the hydrogen ion concentration of the urine. An attempt is made to keep the p_H of the urine between 5 and 5.2. Enteric-coated pills containing about 7½ grains (0.49 Gm.) of ammonium chloride are given three times daily. Fluids are forced; a minimum of 3 liters per day is given. At the same time that the plan for prevention is in operation, the patient is carefully studied to determine a possible cause of the formation of stones.

Oppenheimer and Pollack [15] followed the method of Higgins in an attempt to secure solution of renal calculi by dietetic measures. Twenty-seven patients were placed on the diet for from six to sixteen months; the average period was eleven months. Twenty of the patients maintained a mean urinary p_H of 5.2 or less. In no case was a complete or partial solution of the urinary calculus noted in the roentgenograms, which were made frequently. Five patients showed an increase in size of their renal calculi, and in 1 case a new stone formed while the patient was on the regimen. In the authors' experience the high vitamin, acid

14. McCague, E. J.: A Preliminary Report on a Study of the Problem of Renal and Vesical Calculi that Occur in the Traumatic Group, Tr. Am. A. Genito-Urin. Surgeons **29**:331-336 (May) 1936.

15. Oppenheimer, G. D., and Pollack, Herbert: Attempted Solution of Renal Calculi by Dietetic Measures, J. A. M. A. **108**:349-352 (Jan. 30) 1937.

ash diet did not cause a disappearance or reduction in the size of renal calculi of the alkaline earth type.

Twinem [16] reviewed 314 cases in which operation was performed for nephrolithiasis. The percentage of recurrence after pyelotomy was 20.9, and that after nephrotomy was 28. The greater incidence after nephrotomy agrees with the results of other investigators, but this point should be interpreted with some qualification since the type of operation originally selected depends on the size, shape, number and situation of the stones. The percentage of recurrence after operations for multiple calculi was greater than that after operations for single stones. The frequency of pseudorecurrence is considerable, and to avoid this possibility, roentgenograms should be taken at the time of operation, particularly in cases of multiple and stag-horn calculi.

The low percentage of recurrence in the opposite kidney after both nephrectomy and conservative operation indicates the significance of local factors in the formation of stones. Phosphatic calculi were found to recur more frequently than calculi of other composition except those composed of cystine. In the 10 cases in which stones recurred the kidneys most frequently were either primarily or secondarily infected. The importance of Bacillus proteus in these cases was emphasized. Methods of avoiding postoperative recurrence were outlined. In 56 cases in which the patients have been treated along these lines for the past year and a half, there has been a recurrence in 5.36 per cent. The possibility of the presence of hyperparathyroidism should be investigated, particularly in cases of bilateral and recurring lithiasis.

Frankenthal [17] reported 2 cases in which cystinuria occurred in sisters; in 1 case there were two stones in the renal pelvis; in the other case there was a hereditary atrophy of the macula in the fundus of each eye. Frankenthal debated whether cystinuria should not be considered a definite mark of degeneration, as it often is found in different members of the same family. It is generally admitted today that cystinuria is a pathologic change in the metabolism of amino-acids. Important for diagnosis is a careful daily examination of the urinary sediment in order not to miss the characteristic cystine crystals. Therapy must be as conservative as possible, because the stones recur easily. Diet should be poor in proteins, and large doses of sodium bicarbonate should be administered.

Domanski [18] presented a scheme for the analysis of the components of renal calculi. The system of analysis described did not include a

16. Twinem, F. P.: A Study of Recurrence Following Operations for Nephrolithiasis, J. Urol. **37**:259-267 (Feb.) 1937.

17. Frankenthal, L.: Ueber Cystinurie und Cystinsteine, Arch. f. klin. Chir. **187**:414-424 (Jan.) 1937.

18. Domanski, T. J.: Renal Calculi: A New Method for Qualitative Analysis, J. Urol. **37**:399-406 (March) 1937.

test for every presumably possible component of renal calculi. Specificity and simplicity of procedure were the chief concern. The selected list of substances to be identified were those commonly encountered in the chemical laboratories of the Jersey City Medical Center, and it has been found adequate in dealing with 35 consecutive mixed renal calculi.

The stone is placed on a clean piece of filter paper, moistened with alcohol and scraped free from adherent blood with a scalpel. The specimen is then sawed in half along its shorter axis and examined for the presence of nucleus and stratification. When the latter is present, there may occur a great many layers. Actually, there has never been found a subsequent clearcut chemical difference between more than two alternating varieties. Preparatory to chemical analysis, the stone, or a portion thereof, is ground to a fine powder in a small glass mortar with a glass pestle. Separate layers and nucleus are treated in the same way. The following scheme of analysis is then followed:

To test for uric acid and urates, one should sprinkle a small quantity of the original pulverized sample into a small porcelain casserole. A few drops of concentrated nitric acid should be added and the mixture evaporated to dryness on a water bath. A red color signifies uric acid or urates or both.

Weidel's test, when negative for uric acid, produces a lemon-yellow residue. To test for xanthine, one should add a few drops of a concentrated solution of sodium hydroxide to this residue. In the presence of xanthine a red color is produced.

To test for cystine, one should add from 5 to 10 cc. of ammonium hydroxide (20 per cent) to a small portion of the original pulverized sample, shake vigorously for a few minutes, filter and place a few drops of the filtrate on a clean slide. On the evaporation of ammonia, the characteristic flat hexagonal crystals of cystine can be recognized microscopically under the low power objective.

The presence of phosphates may be determined by placing a small amount of original pulverized sample in a pyrex tube and adding about 5 cc. of ammonium molybdate reagent and an equal volume of concentrated nitric acid. After it is carefully heated to the boiling point, a yellow precipitate appears if there are phosphates.

To test for carbonate, one should add approximately 10 cc. of 10 per cent hydrochloric acid to a portion of pulverized sample. The evolution of gas (carbon dioxide) indicates carbonate (as calcium carbonate).

To test for oxalate, the hydrochloric acid mixture prepared for the carbonate test should be filtered and the filtrate placed in a small casserole. It should be made alkaline to litmus with ammonium hydroxide (15 per cent) and then just acid with acetic acid (5 per cent). After it stands for at least half an hour, it should be centrifugated at high speed until the precipitate, if any, is firmly packed at the bottom

of the tube. Most of the supernatant solution should be decanted, and a drop of the sediment should be placed on a clean slide and examined microscopically under a low power objective for the characteristic crystals of calcium oxalate. The appearance of a crystalline precipitate in the acetic acid solution is indicative of oxalate.

All the stones analyzed by Domanski at the time of his report had been "mixed" stones. In each case more than one substance had been isolated and identified. He did not consider these facts unusual in the light of the medium in which the stones are formed, the possibilities of coprecipitation and the high insolubility of the salts separately considered. The occurrence of phosphates appears universal. Even in the case of a "pure" uric acid stone, appreciable quantities were found. Calcium oxalate almost always accompanies phosphate but usually in relatively smaller quantities; calcium carbonate less frequently accompanies phosphates and most often in quantities small enough to merit the term "impurity."

Tuberculosis.—De Illyès [19] pointed out that urogenital tuberculosis is always secondary to some primary focus elsewhere in the body, which may exist in the form of a cured process and may or may not be detectable. The primary focus most frequently is situated in the lung or in some of the lymph nodes in the hilus or the mesentery. Within the urinary organs themselves, the primary focus is most often the kidney; within the genital organs, it is most often the prostate gland. Both of these are important centers, and there are innumerable modes of propagation. The simultaneous condition of the urinary and genital organs is very common; but of these organs, the kidney is most frequently infected.

At the clinic of the University of Budapest, in 1,071 patients, or 52.4 per cent of 2,043 operated on for purulent infection of the kidney, the condition was tuberculosis. After long study and experimentation, de Illyès concluded that the presence of tubercle bacilli in the urine does not justify the conclusion that there are tuberculous changes in the kidney unless at the same time pus is found in the sediment of the urine. This is a sign of inflammatory reaction caused by the bacillus. To diagnose tuberculosis, the presence in the urine of pus provoked by the Koch bacillus is absolutely necessary. It is known that in the normal kidney the filament of the glomerulus does not allow the tubercle bacillus to pass, but that pathologic changes which arise in the kidney may allow it to penetrate into the urinary tract, or that because of the slowness with which the blood circulates in the filter the tubercle bacillus arriving there may remain so long in contact with the wall as to produce a toxin and lead to tuberculous nephritis and albumin or cylinders in the urine,

19. de Illyès, Géza: La tuberculose urogénitale, J. d'urol. **42**:309-326 (Oct.) 1936.

which are due to degeneration of the renal parenchyma. In the laboratory of de Illyès some of the experiments showed that after the injection of large quantities of tubercle bacilli into the circulation none of these has ever been found in the renal canaliculi in less than one week afterward, that is, not until there had been time for a specific change to occur, as was found by dissection. On the other hand, there were instances in which millions of bacilli had been injected into the circulation for thirty-six days, yet one was never found in the urinary canals after dissection.

In cases of early tuberculosis of the kidney, the exact localization can be found by separate catheterization of the two kidneys, while there is still no change at the vesical orifice of the ureter. In cases in which medical examination does not furnish indubitable proofs of tuberculosis, it is advisable to wait some weeks or even months; this does no harm and will make possible the finding at a later time of proper indications for operation. In every case the moment for establishing such indications should be weighed with great care, since when one removes a kidney one removes the organ which is the most important for the production of antitoxin. One should accordingly not operate too early, even if 4 of 6 patients are going to die from the disease at a later time. Both in early infection and also in advanced infection that has been neglected, and in cases in which tuberculous ulcers have rendered cystoscopy impracticable, diagnosis is difficult; however, in typical infections, such as are seen daily, the Ziehl-Neelsen stain makes diagnosis easy, and pyelography is rarely necessary.

De Illyès has never seen a patient who had renal tuberculosis recover spontaneously, but he has seen a tuberculous kidney become cavernous and caseous, the ureter become thick walled and an autonephrectomy result, so that the patient feels practically well again.

In cases in which the infection is bilateral the author is loath to operate and first tries every possible kind of treatment, including the Gerson diet, injections of the type described by Sailer (iodine-guaiacol-pancreas-lipase), injection of the tubercle bacillus vaccine described by Vaudremère and sometimes roentgen irradiation, which occasionally is favorable.

Of 617 patients who underwent nephrectomy for renal tuberculosis during the fifteen years from 1920 to 1935, 119 were alive and in good condition and 322 were in poor condition. The former were chiefly intellectual persons who were able to live under conditions of material comfort, and the latter were mostly persons who belonged to the laboring class. Twenty-five per cent of the latter were able to resume their work.

The article contains an excellent bibliography.

Mathé [20] reported 6 cases of unilateral renal tuberculosis in children and adolescents. Four of the patients were living and well eight years, six years, eleven months and three months, respectively, after operation. One died six months and 1 died five years after operation, because of generalized dissemination of the disease.

In reviewing 4,698 cases of unilateral renal tuberculosis, Mathé found that 565 (12 per cent) of the patients were from 1 to 20 years of age, 0.42 per cent were from 1 to 5 years, 1.08 per cent were from 5 to 10 years and 10.5 per cent were from 10 to 20 years. Necropsy statistics have demonstrated the incidence to be much higher (from 25 to 30 per cent).

In spite of the invention of a perfected small caliber cystoscope for use with children, the more general use of tests for renal function and the use of the roentgen rays, many pediatricians and physicians still fail to take advantage of these improved and more accurate methods of investigation in cases in which children are suffering from chronic infection of the urinary tract.

Mathé emphasized the importance of bearing renal tuberculosis in mind when making a urologic examination of children who have chronic cystitis, persistent pyuria and relapsing pyelitis.

Although clinical quiescence and autonephrectomy have been overenthusiastically hailed as spontaneous healing of renal tuberculosis, nephrectomy is the treatment of choice for the unilateral type, which affects children as well as adults, and if this operation is performed sufficiently early it is followed by a large percentage of cures.

Lett [21] stated that renal tuberculosis is not a primary and independent disease; it is a local manifestation in a tuberculous subject and is a result of a systemic infection.

In London and the surrounding districts, renal tuberculosis is comparatively rare. An analysis of the records of the London Hospital showed that among 116,713 patients admitted to the surgical wards during the twelve years from 1923 to 1934 there were only 208 who had renal tuberculosis, and in more than a third of these nephrectomy was considered inadvisable on account of their serious condition and the presence of active tuberculosis elsewhere or because the disease was bilateral. In Paris, on the other hand, no less than 568 nephrectomies were performed in Marion's clinic during the ten years from 1919 to 1928. Lett stated that 20 per cent of the patients admitted to urologic clinics suffer from renal tuberculosis and that of a series of 100 consecutive operations on the kidney 50 were performed on patients who were suffering from this disease.

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20. Mathé, C. P.: Juvenile Tuberculosis of the Kidney: Diagnosis and Treatment, Surg., Gynec. & Obst. **63**:283-292 (Sept.) 1936.

21. Lett, Hugh: Renal Tuberculosis: Early Diagnosis and Treatment, Lancet **2**:1313-1318 (Dec. 5) 1936.

The onset of renal tuberculosis is insidious and may be symptomless. Repeated examinations of the urine should be made, especially in every case in which a patient is undergoing treatment for extraurinary tuberculosis.

In some cases early renal tuberculosis can be cured by medical treatment provided that the condition is recognized before there is any alteration in the renal calices.

Unilateral renal tuberculosis becomes a surgical problem as soon as pyelograms show the changes characteristic of the disease, for it is then beyond the hope of cure by medical means alone.

The patient may be free from disturbing symptoms for prolonged periods, but the disease will almost invariably progress and ultimately prove fatal unless the kidney is removed.

Treatment in a sanatorium should always follow nephrectomy and in some cases precede it, as renal tuberculosis is not a primary disease; it is always secondary to a systemic infection and indicates a lowered resistance to the tubercle bacillus. The close cooperation of the general practitioner, the physician and the surgeon is desirable and indeed essential.

Remete [22] reported a series of 30 cases of tuberculosis of the urogenital tract or tuberculous fistulas of long standing after operation. Blood cultures were made for tubercle bacilli by the method of Löwenstein.

He came to the following conclusions: In active tuberculosis of the urogenital tract, tubercle bacilli could never be found in the blood. The same was true in cases in which fistulas followed removal of a tuberculous organ. Tuberculous bacteremia is rare, and its finding does not aid in the early diagnosis of urogenital tuberculosis.

Hydronephrosis.—Solcard and Pervés [23] reported a case of hydronephrotic horseshoe kidney. The patient was a man aged 21 years, who apparently was in good health except for intermittent bilateral pains in the lumbar region and the presence of a soft tumor, which was the size of a mandarin orange (tangarine), situated to the right of the umbilicus and adherent to the deep tissues. Movements of the tumor were restricted vertically, but it could be moved transversely and from within outward; such movement caused pain on both sides in the lumbar region.

The first diagnosis was hydronephrosis of abnormal form on the right side, but catheterization revealed a hydronephrotic horseshoe

22. Remete, Tibor: Zur Frage der Koch-Bazillenzüchtung aus dem Blute bei den tuberkulösen Erkrankungen der Harn- und Geschlechtsorgane, Ztschr. f. urol. Chir. u. Gynäk. **43**:49-57 (Feb.) 1937.

23. Solcard and Pervés: Hydronéphrose infectée, et péritonite mortelle consécutive, dans un cas de rein en fer à cheval, Arch. d. mal. d. reins **10**:291-297 (May) 1936.

kidney which extended in a frontal direction and tended to resemble the "L-shaped kidney" of Papin. Roentgen examination showed that on the right side the catheter followed a normal course, whereas on the left it deviated to the right on reaching the promontory of the sacrum, crossed the vertebral column and stopped a short distance beyond this point. The injection of air revealed a normal pelvis and calices on the right side; on the left, however, there was an enormous pocket which was outlined transversely in front of the vertebral column; 150 cc. of opaque fluid could be injected before the distention became painful. After pyelography, however, a high temperature developed; the urine became purulent and contained streptococci, Bacillus perfringens and a virulent strain of colon bacilli, none of which yielded to treatment with serum. Toxemia was so severe that a transperitoneal nephrectomy could not be performed; therefore, expectant treatment was carried out. It was not until forty days after pyelography that an attempt was made to find the iliac portion of the left ureter, to fix it to the abdominal wall and to dilate it and drain the infected pocket, which was found to contain 100 cc. of thick and fetid pus. Peritonitis developed, and the patient died.

At partial necropsy injection of iodized poppy-seed oil 40 per cent into the arterial system of the kidney showed that the kidney was poorly vascularized. It is certain that a pathologic kidney which has a poor blood supply and is associated with retention and separated from the intestine only by the peritoneum must become infected sooner or later. One may say that the traumatism inflicted by the pyelography facilitated infection; in addition, as the usual bromide solution was not available, the authors injected colloidal silver, which, although theoretically aseptic, is more dangerous. It has been said to cause irritation of the renal pelvis and calices. In reviewing the case, the authors were inclined to believe that if they had performed a transperitoneal nephrectomy (this was the only procedure that seemed to offer any hope of success, owing to the peculiar form of kidney), they might perhaps have saved the patient, or that after infection had closed the peritoneal route against them they should have performed an iliac ureterostomy at once, since a pyelotomy on a median pus pocket surrounded with vessels and already bound down by adhesions was too dangerous. At any rate, they agreed with Tourtou in the opinion "that in congenital malformations one should be much more of an 'interventionist' than in kidneys normally situated."

Astraldi and Repetto [24] reported an unusual case, in which a bilateral hydronephrosis which was due to inferior polar vessels was accom-

24. Astraldi, A., and Repetto, R. L.: Hydronéphrose double par vaisseaux polaires inférieurs avec calculose bilatérale, Arch. d. mal. d. reins **10**:255-267 (May) 1936.

panied by bilateral calculosis. The patient was a girl aged 14. Since about the age of 8 years she had had bilateral pain in the lumbar region. She had had pain in the hypogastrium and several attacks of renal colic; one of these attacks had been associated with hematuria. General malaise and intense asthenia finally had developed. The disproportion between the relatively small size of the stones (one on each side) and the enormous hydronephrotic distention made it seem impossible that the stones were responsible for the latter condition. The etiologic cause of the stones was to be sought rather in the presence of the polar vessels, a congenital anomaly; the pathologic changes, then, were the result and not the cause of the lesion. The bilateral changes pointed definitely to this conclusion.

Therapy naturally had to consist of both medical and surgical procedures and should include treatment of focal infection, local treatment of renal infection, surgical extraction of the calculi, postoperative treatment of the septic foci to prevent the recurrence of calculi and, finally, reduction of the hydronephrosis by section of the polar veins. Retention and infection both had to be taken into account. There was a vicious circle. The retention could not be definitely overcome before operation, as the lesion was congenital and its cause (the polar veins) still existed. While the infection might have been improved, one could not hope to get rid of it altogether while the retention persisted. But the age of the patient and the apparent size of the calculi, as well as the results of the tests of renal function, all indicated operation. The two kidneys were operated on; the interval between the operations was forty-five days. In each instance the polar vessel was sectioned, and a single calculus was removed by inferior pyelotomy. On the basis of their experience in this and other cases, the authors were convinced that in cases in which there is pyelitic dilatation the use of an indwelling catheter after pyelotomy retards cicatrization rather than hastens it as it does in cases in which there is neither dilatation nor retention. The wounds in this case seemingly would not close until the catheter was withdrawn.

In view of the great size of the hydronephrosis, it was not to be expected that there would be any material reduction in the size of the pyelitic sac, and a roentgenogram made ten months after operation confirmed this assumption. The urine still contained staphylococci and pus. A small calculus, the size of a hazelnut, had been eliminated shortly before the report was written, despite the multiplicity of methods employed to combat infection. Postoperative treatment included use of indwelling catheters, lavage of the renal pelvis, administration of vaccines and extirpation of septic foci. It appears to be impossible to acidify the urine, although an attempt is being made to accomplish this by means of a ketogenic diet.

Denervation.—Schneider and Wildbolz,[25] in an effort to elucidate the changes induced by denervation of the kidney and therefore to make the indications for this operation more definite, studied the flow of blood of the normal and of the denervated kidney in dogs. The method used was the thermostromuhr method of Rein. By this method it is possible to measure the flow of blood of unopened blood vessels in situ; in this case the flow in the renal artery and vein was observed under physiologic conditions. The authors found that the normal flow of blood is from 80 to 180 cc. per minute through one kidney of a dog weighing from 9 to 29 Kg., that is, from 4 to 10 cc. per kilogram of body weight. It averages from 1 to 2 cc. per gram of kidney per minute. After they established and registered the normal flow of blood for several hours, the kidney was denervated. For purposes of denervation, both the artery and the vein were carefully freed of visible nerve fibers; chemical sympathectomy was not performed, and the ureter was left intact. The result of this procedure was in all experiments the same. First, they noted a short period of contracture of the blood vessels and the diminished flow of blood followed by a period of unstability and then an increase in the flow; two hours later the flow of blood had risen to 165 to 245 per cent of the original flow. When, at that moment, decapsulation was carried out, the flow of blood increased 20 per cent, probably because of mechanical factors. Decapsulation alone, without denervation, caused a 20 per cent increase in the rate of the flow of blood. The reason for this could not be determined. The most interesting part of the study, however, is the part in which the authors proved beyond doubt that by the simple operative method used the kidney was totally denervated. To prove this they subjected the animal to different well established reactions. The principal one was artificial respiration with air which had a high carbonic acid content. Fleisch has proved by accurate experiments that a rise in the carbon acid content in the blood produces a rise in the flow of blood in denervated regions by dilatation of the blood vessels. Rein was able to show that the contrary was true in regions in which the nerves were intact through vasoconstriction by way of the vasomotor nerve center. Schneider and Wildbolz found that the normal kidney reacted to a high carbonic acid content in the blood by a marked diminution in the flow, notwithstanding a rise in the blood pressure. The contrary was true in a denervated kidney; a high carbonic acid content in the blood was the cause of a marked rise in the flow of blood. Decapsulation alone did not change the reactions of the kidney. In the other physiologic tests used, the carotid sinus

25. Schneider, Max, and Wildbolz, Egon: Dekapsulation und Enervation der Niere und Nierendurchblutung, Ztschr. f. urol. Chir. u. Gynäk. **43**:1-12 (Feb.) 1937.

reflex and the reaction to intravenous injection of epinephrine indicated similar although less clear results.

Nephrofibrosis.—Cumming and Schroeder[26] reviewed 15 cases of renal atrophy and sclerosis and named the condition "nephrofibrosis." They expressed the opinion that even though the lesion in the diseased (atrophic) kidney appears to indicate an old infarct the involvement actually may be the result of ascending infection associated with pyelonephritis, vascular changes and limitation of the nephrofibrotic areas, which correspond to the units supplied by those particular vessels which are end-arteries and have no anastomosis proximal to the glomeruli they supply.

Hinman's theory of counterbalance explains many failures in operations, such as those on advanced hydronephrotic kidneys; yet, as Abeshouse said, if this theory were applied to all clinical cases of unilateral surgical diseases of the kidney in which there are varying degrees of destruction of parenchyma on the involved side, it would seem futile to attempt such procedures as plastic operations for hydronephrosis, pyelolithotomy, nephrolithotomy and renal resection.

One reasonable basis for acquired atrophy and nephrofibrosis is that the peculiar renal status develops as a negative response to natural need; if one kidney has long carried the load of renal function, its fellow gets no biologic urge until sufficient disorder occurs to prevent appreciable recovery. The mechanism of this phenomenon may be a gradual lessening of the blood supply and consequent degenerative vascular changes which prohibit a retro-active improvement.

The authors' interest in this subject has been stimulated by the observation that without anticipated or logical reason certain kidneys simply refuse to function after apparently successful and routine operative intervention. This startling condition often goes unrecognized; the patient and the surgeon remain blissfully ignorant of it until some circumstance brings a wary roentgenologist to recognize rank loss in functional activity, a small renal shadow and contralateral hypertrophy of a kidney, or until the interested surgeon finds, to his chagrin, that the operation has rendered the kidney entirely useless.

Trauma.—Wood,[27] during the period from 1929 to 1935, has treated 25 patients who had an injured kidney. Seven of these were children, 18 were adults, 24 were males and 1 was a female. In 11 cases the right kidney was injured, and in 14 cases the left kidney was injured. Eleven kidneys were ruptured, 2 were lacerated and 12 were contused. Ten injuries were caused by automobile accidents, 8 injuries occurred

26. Cumming, R. E., and Schroeder, C. F.: Renal Atrophy, J. Urol. **37**:407-436 (April) 1937.

27. Wood, A. H.: Diagnosis and Treatment of Trauma to the Kidney, J. Urol. **37**:437-465 (April) 1937.

while the patients were engaged in athletics, 4 injuries were caused by horses and 3 injuries were due to industrial accidents.

Excretory urography was employed in 17 cases, and satisfactory roentgenograms were obtained in all instances on the uninjured side and in 12 cases on the injured side. In 3 cases rupture of the kidney was clearly demonstrated. In the 5 cases in which the roentgenograms were not satisfactory on the injured side, 2 patients required nephrectomy and 3 recovered without operative intervention. In 1 case there was extravasation of the opaque solution into the lower pole of the kidney, and four months later a phenolsulfonphthalein test revealed a 60 per cent decrease in function. Retrograde pyelography was employed in 2 cases. Operation was performed in 13 cases; nephrectomy was done in 10 cases, suture and drainage in 2 cases and exploration and drainage in 1 case.

In these 25 cases there were 3 deaths. Two deaths occurred after operation; one of these was due to an injury of the head and the other to postoperative pneumonia. The third death was due to unrecognized rupture of the kidney and injury to the peritoneum and the mesentery, which were determined at necropsy.

Perinephric Abscess.—Nesbit and Keene [28] discussed perinephric abscess which was associated with bronchial fistula. Sixty cases of perinephric abscess were studied. The average age of the patients was 31 years; the youngest patient was 2 years of age, and the oldest patient was 69. Forty-two per cent were between 20 and 40 years of age. Forty patients, or 66 per cent, were males. The left side was involved 32 times and the right side 27 times. One patient had bilateral abscesses. The average duration of localizing symptoms was three months. Thirty-five patients, or 58 per cent, had had symptoms for one month or less. The longest duration was twenty-two months, and the shortest was one day. The operative mortality was 2 per cent. The total mortality, operative and nonoperative, was 25 per cent. Eleven types of bacterial etiology were found. In 20, or one third, of the 60 cases, the condition was correctly diagnosed within three days after the admission of the patient to the hospital or within three days after the development of symptoms, while the patient was undergoing treatment for some other condition. In 6 cases, or 10 per cent, a perinephrobronchial fistula was present; that is, pus from a perinephric abscess had drained through the diaphragm, pleura and parenchyma of the lung and into a branch of the tracheobronchial tree.

A comparison of the cases in which bronchial fistula developed and the other cases in the series reveals one significant fact: The average duration of localizing symptoms was eight months in the cases in which a fistula developed and three months or less in the uncomplicated

28. Nesbit, R. M., and Keene, C. H.: Perinephric Abscess with Bronchial Fistula, J. Urol. **37**:695-705 (May) 1937.

cases. Nesbit and Keene were of the opinion that involvement of the diaphragm, pleurae and pulmonary parenchyma is a common sequel of chronic perinephric abscess but may not be recognized as such. This is well illustrated in one of their cases in which operation was postponed because of coarse râles and dulness over the base of the homolateral lung, which were interpreted as signs of pneumonia. Analysis of the chain of cause and effect in this condition brought them to the conclusion that the only preventive measure is early establishment of a primary diagnosis of perinephric abscess.

When the diaphragm is involved in a penetrating suppurative process from below, the formation of an adhesive pleuritis at the site of penetration prepares the way for a bronchial fistula. In the absence of adhesion between the visceral and the parietal pleura, empyema results. Treatment of perinephric abscess associated with bronchial fistula is the same as for the uncomplicated abscess. Incision and drainage suffice also for the pulmonary sequelae. Resolution of fever is concomitant with the decrease in sputum. However, if empyema develops, drainage of the pleural cavity is imperative.

Infection.—In the communication by Cabot [29] only blood-borne infections of the kidney were considered, and chiefly those produced by the pus-producing cocci. For many years it has been known that various of these cocci may be found in otherwise normal urine of a patient who has a peripheral infection, such as a boil, a carbuncle, a septic wound or an acute infection of the upper part of the respiratory tract. In order to investigate the latter point more closely, a series of 46 patients who had an acute inflammatory process about the mouth, throat or ear were studied. The group was selected from among patients who had urine normal to ordinary tests and some fever but no signs or symptoms suggesting an infection of the kidney. The urine was carefully obtained, always by catheter from the females. It was studied by smear and culture, the smear being made from urine centrifugated for a considerable period at high speed. Organisms were found in the smear in 32 cases and in the culture in 18 cases.

In a group of 10 apparently normal persons who were used as controls, the smear did not contain any organisms, but in 2 there was a positive culture of streptococci.

It is important to note that the pus-producing cocci appear in the urine early in the disease; they disappear as a rule in a few days, although they occasionally persist for several weeks in an otherwise normal urine.

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29. Cabot, Hugh: Blood Stream Infections of the Kidney, Brit. J. Urol. **8**:233-256 (Sept.) 1936.

The lesions are primarily cortical and medullary. They tend to coalesce with the production of massive abscess, usually show early and marked perinephritis and often result in a perinephric abscess.

Besides the classic methods of diagnosis, stress was laid on the value of roentgenography in the diagnosis of these cases. Clouding or obliteration of the shadow of the psoas muscle is generally discoverable by modern methods in cases of perinephric abscess. Scoliosis is not rare, but is a rather late sign. Limitation of the motion of the diaphragm on the infected side, as studied by the roentgenoscope, is more important than the literature would suggest. Particular emphasis was laid on the value of pyelography both by the excretory and the retrograde method. This will give precise information as to the development of massive abscess by showing deformities of the calices. It will also be of much value in showing displacement of the kidney by perinephric abscess, sometimes at an early period. Stress was laid on the value of limitation of the normal mobility of the kidney due to the early development of perinephritis, often in the absence of perinephric abscess. A series of observations showed diminished visualization of the infected kidney as seen in the excretory urogram. Further study will be necessary to evaluate this sign, since it may be produced either by diminution of the function of the kidney or by the obscuring of the shadow of the renal pelvis by overlying inflammatory tissue.

For the purpose of treatment the cases were divided into four groups. Operation is believed to be the only available method of treatment in cases of fulminating infection.

In cases of acute infection the condition is characterized by chills, high fever, leukocytosis and costomuscular tenderness associated with urine that is normal except for the practically universal presence of cocci obtained by staining the sediment after prolonged centrifugation at high speed. In these cases operation has been generally regarded as almost invariably necessary. The author disagreed with this opinion and stated that in a majority of such cases recovery will take place under medical treatment. A position of "armed watchfulness" should always be maintained, and one should carefully observe the reaction of the patient to the disease, particularly as regards the tendency of the pulse to become more rapid and for anemia to develop. Coupled with this should be frequent observations of the conformation of the renal pelvis both by excretory and by retrograde pyelography. This will enable massive abscess and perinephric abscess to be discovered early.

The third group includes cases of subacute infection in which the picture is less stormy but in which there is clear evidence of a lesion of the kidney. In these cases operation is not indicated unless perinephric abscess develops.

The fourth group consists of borderline cases in which the condition occurs not rarely as a complication of infection of the upper respiratory

passages. The diagnosis can be made only on the basis of slight costomuscular tenderness, fever which is not satisfactorily accounted for by the conditions in the throat and by the finding of cocci in the urine. In these cases operation is practically never necessary, since perinephric abscess is very uncommon.

Indications for operation are: (1) the fulminating type of infection; (2) the widespread infection of the kidney with which the patient seems unable to control the disease, as shown by the development of anemia and failing general conditions; (3) massive abscess of the kidney, as shown by the pyelogram, and (4) perinephric abscess.

The types of operation indicated are: (1) nephrectomy in cases of fulminating infection; (2) nephrectomy for widespread infection without massive abscess, when the patient is losing ground; (3) decapsulation and drainage for a massive abscess which is well localized, and (4) exploration for perinephric abscess. The precise operation is determined by the following factors: (*a*) If the pyelogram shows no evidence of massive abscess or evidence of deformity of the pelvis, simple drainage should be employed; (*b*) if there is perinephric abscess and evidence of massive abscess, the perinephric abscess must be drained and the kidney explored in the region indicated by the pyelogram, and (*c*) if there is perinephric abscess of long standing, even with evidence of destruction of renal tissue as shown by the pyelogram, drainage of the abscess alone is indicated in the first instance. Many of the patients with long-standing abscess will then recover, since the massive destruction of the kidney has already drained into the perinephric space. It is not wise to uncover the kidney widely in such cases, since extensive dissection will be necessary with the breaking down of well established barriers. If after the perinephric abscess has been drained the wound fails to heal, or if it reopens after healing, further exploration can be safely undertaken.

URETER

Anomalies.—Campbell [30] reported 17 cases of ectopic orifice of the ureter; the patients were infants and children. Nine of the patients were examined urologically, and in 7 cases the orifice was seen. Persistent pyuria was the indication for urologic investigation in these 7 cases.

Ectopic orifice of the ureter occurs twice as frequently in females as it does in males. In this series of cases, the anomaly occurred on the left side in 11 cases, on the right side in 4 cases and on both sides in 2 cases.

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30. Campbell, M. F.: Ectopic Ureteral Orifice: A Report of Seventeen Cases in Children, Surg., Gynec. & Obst. **64**:22-29 (Jan.) 1937.

The urinary tract above the ectopic orifice is dilated as a rule. The orifice, however, is usually large, so that urinary obstruction and back pressure are not commonly factors in producing the dilatation. In most cases of ectopic ureter a normal urinary schedule is accompanied by a constant urinary dribbling. This indicates that the external sphincter is inadequate or that the ectopic opening is peripheral to the sphincter. Children as well as adults who have this condition are often treated for enuresis. In cases in which the urinary control is normal, a condition which is less common, the orifice opens behind a competent sphincter. The persistence of disturbances of micturition or of pyuria in either a child or an adult requires a thorough urologic examination. Excretory urography may yield a suggestive clue when ureteral reduplication is revealed, but two isolateral orifices are not found in the bladder, and retrograde pyelography proves that incomplete ureteral reduplication does not exist.

If the ectopic ureter is the only one for the kidney, transplantation to the bladder is indicated in the absence of marked infection. In the presence of significant infection of the anomalous segment and if the condition of the opposite kidneys permits, ureteronephrectomy is the usual indication. If the opening of the ectopic ureter is from one of two reduplicated ureters and only the ectopic segment is diseased, ureteroheminephrectomy should be carried out. If both renal segments are diseased and the opposite kidney is adequate, nephrectomy is the treatment of choice. When nephrectomy alone would leave a large dilated ureter, ureteronephrectomy should be performed. In Campbell's series of cases, ureteroheminephrectomy was done five times, ureteronephrectomy and bilateral ureterostomy each was done once and operation was refused once. The youngest patient to undergo renal resection was 6 months of age. All the patients survived. Four were known to be bacteriologically cured. In another case in which a child was temporarily cured, reinfection subsequently occurred. Two years later, fibrosarcoma of the scapula developed.

Extravesical ureteral openings are reported relatively seldom in the literature. Both sexes are affected, but the condition is usually symptomless when it affects the male. McKirdie and Polkey [31] reported a case in which a ureter opened into the seminal vesicle, and they gave a gross and microscopic description of the specimen. Nineteen similar reports have been found in the literature; these were briefly summarized. A brief résumé of the embryology and a discussion of the accepted theory of causation of the malformation were presented.

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31. McKirdie, Matthew, and Polkey, H. J.: Extravesical Ureteral Opening into the Seminal Vesicle, J. Urol. **37**:706-714 (May) 1937.

Furniss [32] reviewed the reported cases of supernumerary ureter which was associated with an extravesical opening.

In Thom's classification, the most complete of the reviewed published articles, there were 185 cases, 63 being in males and 117 in females; in 5 cases the sex was not mentioned. The supernumerary ureters were on the right side in 82 cases and on the left side in 73 cases; no information regarding the side was given in 22 cases. There were 58 cases of single ureter with extravesical opening, 6 cases of bilateral single ureter with extravesical orifice, 96 cases of unilateral duplication with extravesical opening of accessory ureter, 2 cases of complete duplication of the ureter on one side with extravesical opening of both ureters, 21 cases of bilateral complete duplication with extravesical opening of only one ureter and 2 cases of bilateral duplication of the ureter with extravesical opening of all ureters. In the male patients, the opening was found in the prostatic urethra 33 times, in the seminal vesicles 17 times, in the vas deferens 6 times and in the ejaculatory ducts 5 times. In the female patients, the opening was found in the urethra 37 times, in the vagina 32 times, in the uterus 3 times and in the vestibule 45 times.

Among the females, a history of incontinence since birth was usual. The urethral orifice may have been situated so near the bladder that the urine was returned to the bladder in the waking state and leaked when the child was relaxed in sleep. At times pyuria, for which no satisfactory explanation can be found either in the bladder or in the kidneys the ureters of which drain into the bladder, is the only sign. In some cases fever, without any localizing signs, or pain in the region of the kidneys may be the only expression of the trouble. Intravenous urograms are of value if there is enough functioning tissue in the upper part of the kidney to eliminate the contrast medium, a condition seldom found. Indirectly, they may help if they show absence of the normally visualized upper calix of the kidney and if above this there is a shadow of more kidney tissue than one ordinarily would find with a single renal pelvis. Occasionally, the dilated accessory ureter can be seen cystoscopically, when the ureters run rather superficially under the vesical mucosa. In some instances the dilated ureter has been noted by palpation and inspection on the under surface of the urethra.

Many treatments have been devised. These include ligating the ureter, anastomosing it into the bladder or intestine, uniting the pelves of the two portions of the kidney, heminephrectomy and nephrectomy. Before instituting any plan, the following facts should be determined: the amount of renal tissue drained by the accessory ureter, the degree of distention of the ureter and pelvis, the function of the portions of

32. Furniss, H. D.: Supernumerary Ureters with Extravesical Openings, J. Urol. **37**:341-357 (March) 1937.

the kidney and of the opposite kidney and whether or not there is infection. Furniss expressed the belief that any form of simple ligation should be abandoned. Anastomosis to the bladder should be done only when the ureter is that of a solitary kidney or is the only ureter draining one of a pair of kidneys, or in cases in which a single ectopic ureter drains both kidneys or in which one of a pair of ureters drains a worth-while portion of a kidney in which there is little, if any, infection. Furniss stated that the best and most satisfactory method is heminephrectomy. This usually can be done rather easily. The upper and lower poles nearly always have an independent blood supply; the upper pole is most often demarcated from the lower pole, and the separation can be made with a minimal loss of blood. Even when the two portions appear to be intimately blended, the upper can be cut away with the electric cutting instrument. Individual bleeding points can be caught with the forceps and ligated or electrocoagulated. A technical point of advantage is to strip the capsule from the upper pole toward the lower pole before resection. This helps in closing the wound in the upper pole. If there is oozing, fat or, even better, muscle tissue can be placed in the wound and the capsule sutured over it.

Stone.—Moore [33] discussed the factors which determine the management of ureteral stones and described an improved method for their cystoscopic removal.

The larger the stone, the less likely are manipulative efforts to succeed. If the calculus is more than 1 cm. in diameter and if it seems probable that more than three cystoscopic treatments or ureteral dilations will be required before the stone can be recovered, it is often less trying on the patient and possibly less hazardous, to proceed immediately with an extraperitoneal ureterolithotomy. A stone impacted in the lower third of the ureter is decidedly more amenable to manipulative removal; if it is lodged in the upper or middle third and is not more than 1 cm. in diameter, one may await its passage into the more favorable pelvic portion. In the case of larger stones, lumbar ureterotomy should be more frequently employed. One hundred and thirteen cases form the basis of this report. In 20 cases the stone was lodged in the upper part of the ureter; in 13 of these cases cystoscopic treatment was not attempted; of the 6 cases in which manipulation was tried the results were successful in only 2. Seventeen patients were subjected to lumbar ureterolithotomy, and 1 patient passed a small stone spontaneously after cystoscopic examination. On the other hand, of 100 stones lodged in the lower portion of the ureter, 14 were passed spontaneously after the examination. Of the remaining 86 patients, 77

33. Moore, T. D.: Factors Determining the Management of Ureteral Stones: An Improved Method of Their Cystoscopic Removal, J. Urol. **37**:1-11 (Jan.) 1937.

were subjected to cystoscopic treatments, and 64 stones were recovered. Surgical intervention became necessary in 13 cases after manipulation failed; ureterolithotomy was performed in 6 cases, without previous efforts to remove the stone instrumentally; in 3 cases a nephroureterectomy was performed because of complete renal atrophy which was secondary to a long-standing obstruction due to calculus.

Duration of symptoms always merits consideration; if the onset is recent, expectant treatment for a few days may be followed by the spontaneous passage of a stone. If there is a history of attacks of renal colic occurring over a period of weeks or months, it is obvious that the assistance of the urologist will be necessary.

Urinary stasis of varying degrees is a feature demanding special consideration in many cases and is most safely demonstrated by excretory urography. Stasis must be recognized as fertile soil for infection; it occasionally prohibits the employment of manipulative methods. The function of the involved kidney is of importance. In this regard an excretory urogram may be misleading; temporary or reflex inhibition of function, with absence of the medium on the affected side, is of frequent occurrence in the presence of ureteral stone. In a recent study of 150 urograms there were 11 in which one or more stones in the ureter caused absence of function which was temporary, as proved by return to normal function after removal of the calculi. If no function is demonstrable either by urography or by the dye tests, nephrectomy may be indicated.

Females withstand manipulation better than males. If multiple catheters are used, more of them may be passed at one sitting by reintroducing the cystoscope alongside catheters previously inserted. On women, vaginal ureterolithotomy may also be employed advantageously. Elderly and enfeebled patients require the most gentle treatment possible and should not be subjected to a long series of instrumentations; only when the stone is small and unquestionably favorable for manipulative removal should this type of treatment be instituted. In infants and children ureteral stones fortunately are comparatively rare and are usually of small size. The mere passage of a catheter may bring about expulsion of the stone. In this series of 113 patients the only death was that of a man aged 70, whose general condition was unfavorable for any type of treatment. He was obese and had diabetes, hypertension and chronic myocardial degeneration.

McKay described an ingenious dislodger, embodying the principle of multiple catheters but using, instead, four long silk threads attached to a small bougie (no. 3 French) of whalebone. He suggested that it be passed beyond the stone, after the ureter was thoroughly dilated, and by pulling on the threads and holding the filiform bougie firmly it would become slightly bowed; the stone would become entrapped by

twisting and could be brought down into the bladder by traction. After Moore had tried this technic in a few cases with indifferent success, it occurred to him that this device might be used to a better advantage by passing the filiform which carried the threads well beyond the stone, even as far as the renal pelvis, and then withdrawing it by traction only, with no effort to induce bowing. The obstructing stone causes the threads to wad above it and form a small mass, which does not easily permit the point of attachment of the threads to be withdrawn unless the stone also is brought along. If a calculus is engaged in this way and does not slip out easily by steady or intermittent traction, or if for any reason one desires to abandon the effort, the bougie may be inserted further and the strands of thread may be easily drawn out along with the filiform. Since the author has used this technic there have been no accidents, embarrassing experiences or apprehensive moments. It cannot be overemphasized that the ureter should be dilated well before insertion of the dislodger. A direct cystoscope of the Braasch type is preferred, as the catheter guide of this instrument renders unnecessary the passage of the bougie through a length of catheter; moreover, there is no deflector to become entangled with the threads.

This method has been used in a group of 31 cases, in which there were 33 ureteral stones to be removed. Twenty-four stones were removed on the cystoscopic table. Twelve stones were removed at the first treatment; 7 required two treatments, 4 required three treatments and one required four treatments, making a total of forty-two treatments, or an average of one and three-fourths for each stone. When more than one cystoscopy was necessary, an interval of from five to ten days was customary, with gradually increasing dilation of the ureter as a preliminary measure. A few cubic centimeters of sterile oil should be injected before the dislodger is passed.

Transplantation.—Hinman and Weyrauch [34] said that a study of the literature on uretero-intestinal implantation leaves one with a feeling of disappointment at the lack of improvement with the advent of newer methods and greater experience. It would seem that every surgical principle imaginable has been tried. Of the 11 listed by Hinman and Weyrauch in their study, the submucosal principle has a distinct advantage in theory and has shown the best results in practice. Nearly all modern technics make use of it, either as the primary or as the secondary principle. Even the newest methods which utilize the intact ureter with the idea of asepsis are primarily submucosal. The problem of the formation of an opening between the ureter and the bowel at the second stage of the operation, by methods based on the eleventh principle,

34. Hinman, Frank, and Weyrauch, H. M., Jr.: A Critical Study of the Different Principles of Surgery Which Have Been Used in Uretero-Intestinal Implantation, Tr. Am. A. Genito-Urin. Surgeons **29**:15-156 (May) 1936.

has not been satisfactorily solved. The final test of a successful implantation is not only recovery from the operation but survival with normal ureters and kidneys. The lateral opening of those technics using it is unsatisfactory because of its tendency to constrict and produce obstruction. In practice, the advantage gained by the healing of the ureter in its intestinal channel under conditions of asepsis is offset by the difficulties of establishing a full opening afterward. Simpler one stage methods which give an orifice at the end of the ureter may prove superior.

In addition to these elementary principles representing the 11 major differences in technic, several conditions must be recognized as common to all technics, no matter how elementary or compound. These are the basic principles of intestinal and ureteral surgical procedures and must be fulfilled by any method. What produces localized necrosis of the intestine or perforation and tearing out of sutures, the common causes of postoperative leakage and peritonitis? What produces the constriction which leads to intestinal obstruction? Why is anemic infarction, extensive necrosis, diffuse ureteritis or marked dilatation of the ureter seen at necropsy? Too often the answer is failure to follow the simple well known rules for intestinal and ureteral surgical procedures. The only layers which are safe for suturing are the submucosal layer of the bowel and the adventitia of the ureter. Sutures cannot penetrate the lumen of either without danger. Their blood supply cannot be disturbed to any great extent. Neither one can be unduly traumatized, twisted or displaced. These are some of the basic principles that must be followed.

The marked difference between the risk of implantation for exstrophy (less than 15 per cent) and that for cancer (almost 50 per cent) arises partly from differences in the age period but mostly from the added risk of the operation for the malignant condition. Statistics also show that implantation in two stages (one ureter at each), as done for most exstrophies, is safer than simultaneous bilateral implantation. The latter is the usual method used for malignant conditions, because of the necessity for a second operation to remove the cancer. Perfection of the principle of the intact ureter to a one stage operation or the development of any safe procedure in one stage will lower the mortality of cystectomy for malignant conditions. Until such a procedure is developed, the implantation of the second ureter at the time of cystectomy is the safest plan.

The theoretical advantage of extraperitoneal operations has not proved to be practical. Peritonitis results from leakage after operation and not from contamination at the time. When sutures are placed properly, leakage does not occur. The little protection against leakage gained by extraperitoneal exposure is more than offset by the increased difficulty of implanting the ureter untwisted, unkinked and unobstructed.

Finally, it appears from this study that the problem remains unsolved, and one must recognize that the problem involved is not solely one of surgical technic. There is the unknown, indeterminate but ever present factor of urinary sepsis. Susceptibility to infection varies with persons as does the conjunction at operation of accessory and other factors which favor it. Varying degrees of pyelonephritis and infected hydronephrosis often are present already, and experience shows that such conditions are rather favorable than otherwise when they have led to a well established immunity. Whether the tract above is clean or not, there is the chance that an acute infection will ascend from the bowel to the kidneys as soon as a communication is established. The surgeon who knows the individualistic, technical and bacteriologic factors of success and failure is in a position to reduce the uncertainty of the operation to a minimum. The success of the future may lie along lines of immunity as much as of technic.

Higgins [35] stated that in a series of 78 cases in which transplantation of the ureters into the rectosigmoid was performed, the best results were obtained when the following principles were carried out: In cases in which cystectomy and transplantation are performed for carcinoma of the bladder (modified for children requiring a similar operation), a liquid, nonresidue diet is supplemented by dextrose, candy and gelatin. One thousand cubic centimeters of a 10 per cent solution of dextrose is given intravenously each day. Dextrose is administered in physiologic solution of sodium chloride if the renal function is not impaired; if the function is decreased, dextrose is given in distilled water. Magnesium sulfate is given daily by mouth. One ounce (32 Gm.) of magnesium sulfate is dissolved in 8 ounces (240 cc.) of water, and 1 ounce (30 cc.) of this mixture is given every half-hour, starting at 7 a. m. No magnesium sulfate is given on the day prior to operation. Daily cleansing enemas are given until they return clear. These are given in the afternoon. Two drachms (8 cc.) of camphorated tincture of opium U. S. P. is given at two hour intervals for three doses—usually at 4, 6 and 8 p. m. —the night before operation.

The type of operation to be performed is influenced by the judgment of the surgeon and his familiarity with the various operative procedures. Certainly the operation which is attended by the lowest mortality and morbidity in his hands should be employed.

After operation the patient is maintained in a flat position for eight hours if spinal anesthesia has been employed; then Fowler's position is used. After spinal anesthesia the blood pressure is checked every fifteen minutes until it returns to normal. No food or water is given

35. Higgins, C. C.: Transplantation of the Ureters into the Rectosigmoid: An Experimental and Clinical Consideration, J. Urol. **37**:90-105 (Jan.) 1937.

by mouth for at least forty-eight hours; then small amounts of water are given by mouth for twelve hours. During the following twelve hours administration of clear fluids is started; 1 ounce (30 cc.) is given hourly, and this amount is gradually increased. A low residue diet is then used for from five to seven days. The fluid intake is kept at 3,000 cc. daily. During the first two days, 2,000 cc. of a 10 per cent solution of dextrose is given intravenously and an infusion of 1,000 cc. of saline solution is given daily. After this, fluids are given intravenously, as indicated by the chemical composition of the blood and by the fluid intake by mouth. Narcotics are prescribed as necessary. A rectal tube is inserted at the time of operation. This may be changed but is replaced and left in the rectum from eight to ten days. The rectal tube is gently irrigated with physiologic solution of sodium chloride at intervals of two hours to insure patency. If the transfixion suture technic is employed, gentle traction is made on the rectal tube at the end of twenty-four and of thirty-six hours or until the tube containing the suture is expelled. A plain rectal tube then is reinserted for from five to seven days. The composition of the blood is studied daily. Ten cubic centimeters of methenamine-acetylaminosalicylic acid is given with the dextrose as a urinary antiseptic, for the first three or four postoperative days.

The patients who have benign conditions and who survive the operation—and the ratio of these patients is steadily increasing—may be expected to live an indefinite period. Higgins has seen patients who are living and in good physical condition after eighteen, sixteen and fourteen years, respectively. It is yet too soon to know what the results may be in cases in which patients have been operated on for malignant conditions, but Higgins was certain that it will be possible to salvage a fair percentage of these unfortunate persons who are destined to a miserable end from malignant tumors of the urinary bladder.

Drainage.—Foulds [36] stated that prolonged drainage of the lower part of the urinary tract has long been accepted as a valuable procedure in obstruction of the lower part of the urinary tract. Drainage of the kidney by nephrostomy after the removal of renal calculi is advisable in certain cases as a means of promoting better function and decreasing infection. The use of the indwelling ureteral catheter is recognized in the treatment of pyelitis of pregnancy and other infections of the upper part of the urinary tract. In cases of impacted stones in the ureter and infection in the kidney and dilatation of the ureter, temporary ureterostomy with a T tube offers a means of providing sufficiently prolonged

36. Foulds, G. S.: Prolonged Drainage of the Ureter, Tr. Am. A. Genito-Urin. Surgeons **29**:1-5 (May) 1936.

drainage of the ureter to allow improvement in renal function to occur and renal infection to subside. The T tube is of use in the reconstruction of the ureter after operation for impassable stricture.

Di Maio,[37] on the basis of a special study of 68 cases in which the indwelling ureteral catheter was used, concluded that there are practically no contraindications to its use and that the method not only gives symptomatic relief but conduces to cure in various pathologic conditions of the urinary system. By analyzing his results according to the type of case, he reached the following conclusions:

For ureteropyelitis from various causes (movable kidney, ureteral stone expelled without operative measures), when there is still sufficient tonus of the pelvis and the ureter, endoscopic ureteral drainage results in rapid and certain cure. In cases of abscessed kidney which is not easily distinguished from simple pyelitis, the negative action of this drainage constitutes a means of differential diagnosis, on the basis of which it is possible before the condition becomes too grave to do a conservative operation (decapsulation or lumbar drainage), which can be followed by a radical procedure at a later time. In cases of movable kidney, the hematuria of hydronephrosis ceases with the placing of an indwelling catheter. In cases of pyelitis associated with pyonephrosis of the other kidney and in mild hydronephrosis of one kidney with grave hydronephrosis of the other kidney, ureteropyelic drainage of one or both sides improves the general condition and by curing the pyelitis or improving the mild hydronephrosis makes it possible later to carry out a radical operation on the other side. In cases of hydro-ureteropyonephrosis from ureteral calculus, such drainage (when it can be accomplished) reduces the septic condition and favors a normal postoperative course. Even with inoperable conditions, such as bilateral calculous hydropyonephrosis, it makes some improvement possible, as it does also with hydropyo-ureteronephrosis of gynecologic origin, for which operation is contraindicated.

Even when such drainage is maintained with the same catheter for one to seven days and repeated several times and for many months, it is impossible to establish precise limits or contraindications to its use, since it is possible to apply conservative drainage even in the presence of grave suppurative and inoperable conditions, for the relief it brings. With less severe inflammatory processes of the ureter and pelvis, this drainage can be applied when other common measures are shown to be inefficacious.

37. di Maio, Giorgio: Il drenaggio uretero-pielico endoscopico in alcune affezioni chirurgische settiche uretero-pielo-renali, Arch. ital. di urol. **13**:391-424 (July) 1936.

(To be concluded)